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AUDITORY ACUITY IN HYPERCHOLESTEREMIA.*

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Cholesterol is an unsaturated secondary alcohol existing in the body in two forms[1]: a free form in the brain and red blood corpuscles, and in a form combined with the higher fatty acids as an ester in all other organs, including the blood plasma[2]. Bailey[3] states that cholesterol is formed by the liver and excreted with the bile into the intestines, where it is almost completely resorbed. Chamberlain[4] ventures the opinion that it is synthesized in the suprarenal glands. This endogenous nature of the cholesterol metabolism is firmly established and generally accepted[5]. As further proof of its endogenous origin it is pointed out that starvation is frequently associated with an increase of blood cholesterol.

The cholesterol content of the blood varies within normal limits in man from 140 to 180 m.g. per 100 c.c. A small rise beyond that figure can be obtained[5] after the ingestion of a meal rich in cholesterol but adjustment to normal level soon occurs. In cases of disfunction of the thyroid, pancreas and other endocrine disturbances, alteration of the amount of blood cholesterol occurs. Pregnancy is associated with an increase in cholesterol. Consequently, the choles-

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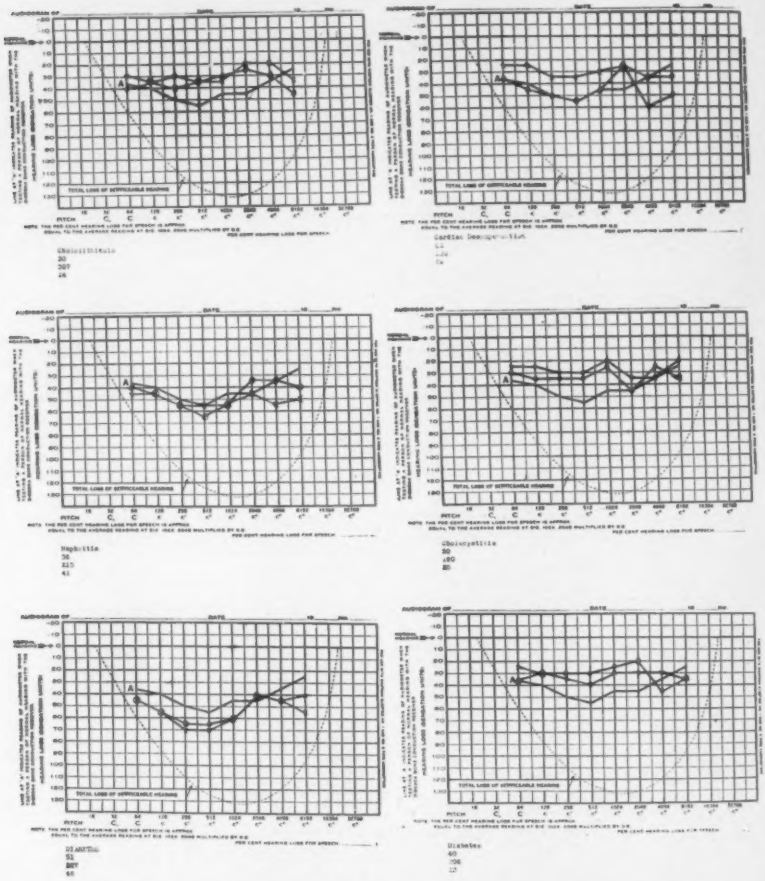
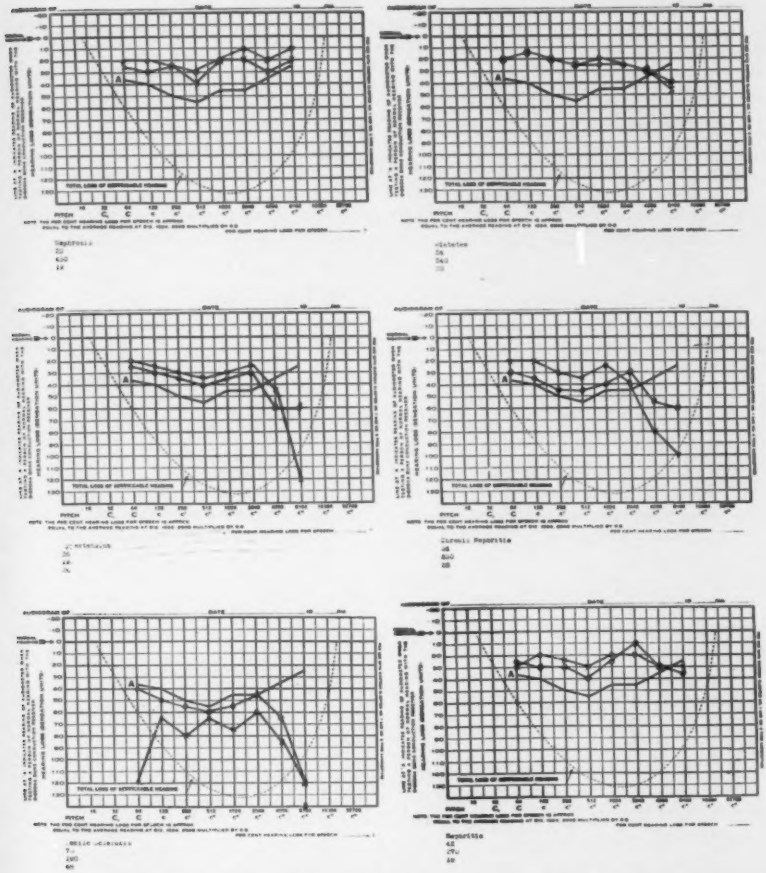
terol level in man is independent of the amount in the ingested food and if the exogenous source falls there are endogenous synthetic sources that maintain the cholesterol at a more or less fixed level in the blood in health.



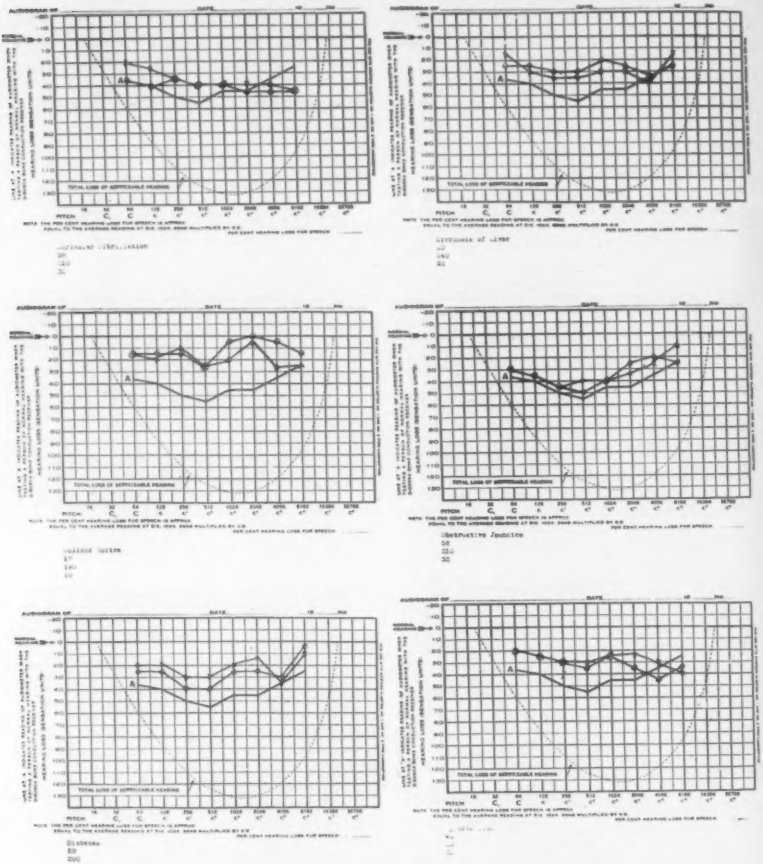
The exact mechanism regulating the cholesterol metabolism is not known but probably has a relation to internal secretion. Pardeu reports a definite increase of blood cholesterol in thyroidectomized dogs. In these the cholesterol level can be reduced at will by feeding the dogs thyroxin. That thyroxin has a definite effect in lowering

the blood cholesterol level is further demonstrated[1] by the uniform finding of low blood cholesterol values in hyperthyroid dogs. Hypocholesteremia also occurs at the height of fever[2] and in a variety of acute infective conditions.



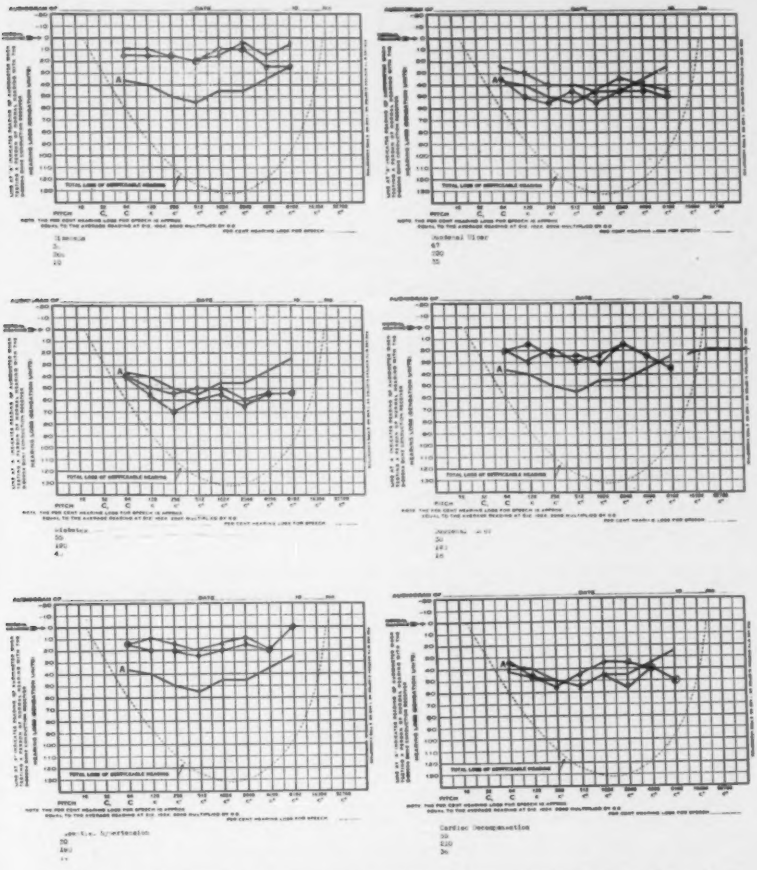
Fineberg[7], in 1932, advanced the theory that cholesterol metabolism is the connecting link in the chain formed by essential hypertension, arteriosclerosis, apoplexy and chronic nephritis. There is a clearly marked hypercholesteremia in all these conditions. He proceeded to show that an increased cholesterol content of the blood

produced changes which are common in these conditions. Applied to the auditory mechanism, he states that the early symptoms of deafness depend on functional changes in the labyrinthine vessels, but after the condition has been of long standing it is possible to histologically demonstrate a continuous contractile condition of the



blood vessels, together with a cholesterol deposit. Histological examination, by him, of temporal bones in 19 cases of hypercholesteremia revealed uniform internal ear pathology. There was a typical lipoidal infiltration in the drum membrane, in the ossicular chain joints, in the basilar membrane, in the ductus cochlearis, ligamentum spirale,

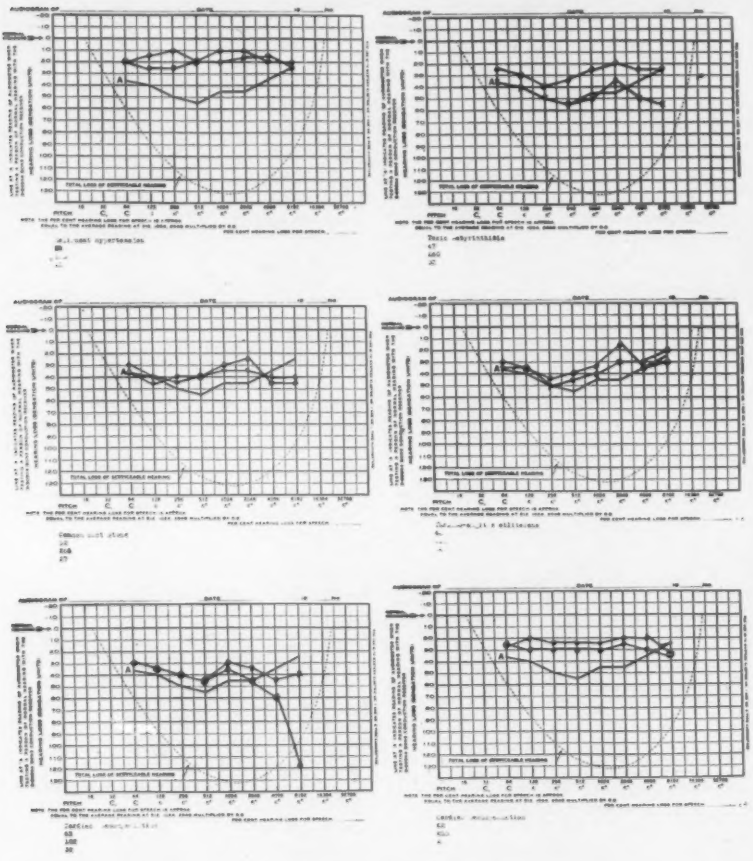
in the epithelium of the striae vascularis, in the membrane vestibular, in the cells of Hensen and in advanced cases in the cells of Claudius. Arteries and capillaries showed changes similar to arteriosclerotic changes. Fineberg concluded that the increased cholesterol content of the blood produced lipoidal changes in the peripheral



auditory end organ in man. He produced hypercholesteremia in rabbits experimentally and showed practically the same histopathological changes.

It was our purpose to establish whether or not the lipoidal infiltration of the ear end organ in hypercholesteremia, as reported by Fine-

berg, resulted in a diminution of auditory acuity. It had been observed that a number of systemic diseases, such as hypertension, nephritis, diabetes, arteriosclerosis, were often associated with a progressive nerve deafness. This group was also characterized by a frequent hypercholesteremia[7]. Was there an association in these



patients between the hypercholesteremia and the deafness found in life, and the lipoidal infiltration found at autopsy? To determine this, we selected for hearing examination 30 medical ward patients on whose laboratory charts we found recorded high blood cholesterol figures. These cases included a variety of diseases of the type exam-

ined postmortem by Fineberg. The ages of the subject group ranged from 17 to 61 years. In only one instance did a patient complain of a partial deafness, in a case of toxic labyrinthitis.

I will now project the audiograms of our 30 cases* on the screen and will ask you to observe particularly the three figures, the age of the patient, the cholesterol level and the hearing loss in percentage.

Tabulating these findings, we established absolute hearing losses in the different age groups, ranging from 16 to 33 per cent, as follows:

Age Group	*% Loss Hearing*
20-29	16
30-39	24
40-49	26
50-59	33
60-69	32

How do these findings compare with those in normal nonhypercholesteremic cases of equal age?

In 1929, C. C. Bunch, from an audiometric study of 353 nonotological patients, reported hearing losses in successive decades to be as follows (percentage computation by us from Bunch's scattergrams):

Age Group	*% Loss Hearing*
20-29	13
30-39	12
40-49	18
50-59	18
60-69	24

Comparing our findings with those of Bunch, we find that there is a loss of hearing over normal loss in hypercholesteremic cases which progressively increases in successive decades from 3 to 15 per cent, as is shown by the last group of figures:

Age Group	*% Normal Loss*	*% Loss in Cholesteremia*
20-29	13	16
30-39	13	24
40-49	18	26
50-59	18	33
60-69	24	32

Are we justified in assuming that the hearing loss in our patients is the result of changes in the auditory end organ caused by high blood cholesterol? Are not other factors, such as the disease process itself, or the state of debility of the patient involved as contributory or causative agents? We again cite figures from a subsequent paper

*Of these 30 cases, 22 were definitely hypercholesteremic and 8 were classified as high normal.

of C. C. Bunch[9], who reports the finding of an almost uniform average loss of hearing of 16 per cent in a group of 251 cases of syphilis, malignancy, arteriosclerosis, cardiac and hypertension of all ages.

From the foregoing it is obvious that systemic disease of itself is not the etiological factor in the production of marked hearing loss. But, in our cases, systemic disease of the type cited above, but accompanied by a hypercholesteremia, did yield on examination constant progressive hearing loss, increasing with age. It may, therefore, be logically concluded that variation in auditory acuity with progressive loss of hearing is attributable to hypercholesteremia with its attendant histological changes in the small group of systemic diseases studied.

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THE SIGNIFICANCE OF BLOOD CHOLESTEROL IN ATROPHIC RHINITIS.

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The theories which have been advanced for the explanation of atrophic rhinitis are numerous. On reviewing these, one notices that the earlier authors have treated atrophic rhinitis as a local disease. They have tried to explain the pathology in the nose and throat by local insults. Examples for an etiological explanation of this type are the specific bacillar theory and Gruenwald's herd-infection theory.

The pathology observed in atrophic rhinitis and in ozena is very extensive. It occurs not only in the nasal chambers and the nasal accessory sinuses but also in the pharynx, larynx, trachea and bronchi[1]. It is not confined to the mucous membrane and submucosa but involves the underlying bone and cartilage. This extent of the pathological process leads to the assumption that atrophic rhinitis is not a local process but the local expression of a general constitutional disease. Among the etiological factors for atrophic rhinitis which have been mentioned in the literature are some which confirm this assumption.

Atrophic rhinitis in the simple and in the fetid form is known to occur in families through many generations. There are several such family trees in the literature. At present I am treating two brothers with ozena, and a family in which the mother and her four children show more or less marked degrees of atrophy of the nasal chambers. The hereditary and familiar occurrence of atrophic rhinitis speaks strongly for the constitutional etiology. To explain it on the basis of infection through contact of the members of the family is not plausible; first, because atrophic rhinitis does not always befall all members of the family, even when they have been in close contact with each other and, second, because inoculation of the nose with human ozena material has failed to produce ozena in animals, as well as in human beings.

In a great percentage of cases of atrophic rhinitis an underdevelopment of the facial skeleton[2] has been observed, which manifests itself in a disproportion between the length and the width of the nasal fossa; they have been found to be wider and shorter than in normal individuals. This more or less constant lack of development of the facial skeleton could be explained as a constitutional anomaly.

Ozena usually manifests itself about puberty; in female patients an aggravation of the nasal condition is often observed at menstrua-

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tion. These facts make a causal connection between atrophic rhinitis and the endocrine glands, especially the sexual glands, probable. Yet authors who have subjected patients with ozena to an examination of the endocrine apparatus and have found disturbances of the same in a great number of the group have not established a definite relationship between these[3].

The sympathetic and parasympathetic nervous system[4] has often been the object of investigation for an explanation of atrophic rhinitis. A general hypotony of both was found by Wiskowsky[5].

Glasscheib[6], who blames lack of vitamin for the production of atrophic rhinitis, has tried to show dystrophic disturbances in the glandular apparatus of the intestinal as well as of the respiratory tract.

Disturbances in the development of the ectoderm, like hypodontosis, hypotrichosis, hypohydrosis and albinism, are occasional findings in patients with ozena and are used to support the assumption of a constitutional etiology.

Fleischmann[7], led by the belief that if atrophic rhinitis is a constitutional disorder, it should evidence itself in abnormal blood chemistry findings, examined the blood of 12 patients with simple atrophic rhinitis and 22 patients with ozena. Chlorin, potassium, calcium, magnesium and blood sugar showed normal values. Of the lipoids, lecithin showed normal values, while the cholesterol content was found to be low throughout. In the group of ozena patients, his values ranged from 10 m.g. per 100 c.c. to 125 m.g. per 100 c.c., meaning an average of 86 m.g. per 100 c.c. In the simple atrophic group, he recorded values from 25 m.g. per 100 c.c. to 130 m.g. per 100 c.c., or an average of 92.6 m.g. per 100 c.c. Taking the normal cholesterol in the blood serum as between 140 and 180, as determined by the method of Authenrieth and Funk, we find that in Fleischmann's findings the values never reach the low normal. Fleischmann grouped his patients in regards to age, sex, duration of symptoms, the degree of atrophy, crust formation and fetor and found that in ozena the lower cholesterol values corresponded to the more recent and the higher values to the more advanced cases. In simple atrophy the opposite is the rule, in that the higher cholesterol content is found in mild and the lower cholesterol level in the advanced cases. Continuous low cholesterol in the blood serum is extremely rare in human pathology. Fleischmann's values are to be understood as constant because comparative repetition of the blood chemistry in some of his patients confirmed the previous results. In order to establish the specificity of low blood cholesterol for atrophic rhinitis, six cases

of chronic hypotrophic rhinitis with polyposis were examined. All showed normal values.

Having found a low blood cholesterol, Fleischmann then proceeded to elaborate upon his theory by explaining the etiological significance and the symptoms of atrophic rhinitis with his findings.

Cholesterol and lecithin are largely responsible for the permeability of the cell membrane. One has the diametrically opposite effect to the other upon water retention, electric isolation and ionization. A disturbance of their equilibrium, as takes place in these cases in favor of the lecithin, will bring about a disturbance in metabolism and affect the tissues. Lecithin is hydrophilic, cholesterol hydrophobic. Excess lecithin not only increases the permeability of the cell membrane but, as Fleischmann assumes, also increases the secretion of the glands, a conclusion which is not permissible *per se*. The fact that the secretion lessens with the progress of ozena seems to him in accord with the increase of cholesterol in advanced cases of long duration.

The chronicity of the inflammatory process is explained by the fact that lack of cholesterol causes a vagotony, which in turn moves the acid-base balance to the sour side, a factor which is supposed to increase susceptibility to infection.

Again the positive lecithin balance is used to explain the atrophy. Cholesterol retards, lecithin increases tissue respiration. Increased oxidation of the nasal mucosa results in atrophy and, as the nasal chamber widens and greater quantities of air pass through it, causes the progress of the atrophic process.

In regard to the crust formation, Fleischmann sees a connection between their high lipoid content and the disturbed lipoid metabolism. The scabs could be looked upon as the cast-off products of oxidation comparable to the scabs caused by artificial oxidation with caustics. He observed scab formation, but without fetor, in a laborer who in his work was constantly exposed to chromic acid fumes.

The question whether the cholesterol deficit is of bacterial-toxic origin is refuted on the basis that chronic infections elsewhere in the body never cause a lowering of the cholesterol level in the blood except in cachectic diseases, and then only to a small degree.

Fleischmann sees in the low cholesterol content of the blood serum a definite constitutional anomaly and supports his theory with the etiological factors which have been mentioned in the beginning of this paper, especially the hereditary occurrence and the congenital lack of development of other ectodermal structures.

As for the practical evaluation of Fleischmann's theory, the surgical narrowing of the nasal chamber is advocated to combat the

noxious oxidation of the tissues. He uses the cholesterol content of the blood as a prognostic index, having observed that better results from the operation correspond to the higher cholesterol values before operation. In the cases which show a low cholesterol before operation, he advises postoperative injections of Helpin, which is a lipoid emulsion containing 10 per cent lecithin, 17 per cent glycerin and 0.9 per cent electrolytes.

Trying to make use of this suggested prognostic aid in my own surgical therapy for ozena, I have examined the blood cholesterol in 15 ozena patients and 13 patients with atrophic rhinitis. I omitted all cases which left doubt as to the diagnosis, particularly definite chronic purulencies of the nasal accessory sinuses and cases with postoperative atrophy, and kept strictly to Fleischmann's method of examination. The cholesterol estimates were done in the Biochemical Laboratory of Beth Israel Hospital under the supervision of Dr. Ella H. Fishberg, to whom I wish to express my gratitude for her kind interest and co-operation.

TABLE I—SIMPLE ATROPHIC RHINITIS.

Name	*Age*	*Duration*	*Sex*	*Atrophy*	*Crusts*	*Chol. m.g. %*
Ida S.	49	20	F	4+	1+	110
Bertha S.	16	1	F	2+	1+	130
Wolf N.	43	25	M	3+	1+	155
Tillie K.	50	30	F	2+	2+	160
Max B.	12	4	M	2+	1+	160
Molly B.	47	20	F	3+	2+	160
Beatrice B.	9	1	F	2+	1+	173
Ida G.	26	5	F	2+	1+	181
Morris B.	13	1	M	2+	3+	181
Joan A.	26	15	F	3+	1+	185
Pauline K.	27	13	F	3+	3+	190
Nettie B.	50	26	F	2+	1+	200
Tillie L.	37	5	F	4+	2+	210

TABLE II—OZENA.

Name	*Age*	*Duration*	*Sex*	*Atrophy*	*Crusts*	*Fetor*	*Chol. m.g. %*
Anna C.	20	5	F	2+	4+	3+	116
John D.	18	3	M	3+	3+	4+	117
Dora L.	38	20	F	2+	2+	3+	152
William D.	25	6	M	4+	3+	3+	163
Mary D.	22	9	F	2+	2+	1+	170
Eva M.	29	15	F	4+	3+	2+	175
Florence T.	35	10	F	2+	3+	1+	176
Lena H.	37	12	F	3+	2+	2+	181
James M.	32	20	M	3+	3+	3+	181
Sadie W.	23	10	F	3+	4+	3+	183
Bridget M.	49	15	F	3+	3+	2+	192
Franco R.	24	5	M	3+	2+	1+	202
Gertrude Z.	28	11	F	3+	4+	2+	210
Richard M.	35	12	M	2+	2+	1+	210
Harriet G.	23	10	F	3+	3+	3+	235

My values for the ozena group ranged from 116 m.g. per 100 c.c. to 235 m.g. per 100 c.c., with an average of 177.5 m.g. per 100 c.c., and in the atrophic rhinitis patients from 110 m.g. per 100 c.c. to 210 m.g. per 100 c.c., or an average of 168.8 m.g. per 100 c.c. As mentioned above, Fleischmann's highest values never reached the normal level of 140 m.g. per 100 c.c. In the 28 cases of both groups which I have examined, 10 showed normal values between 140 m.g. per 100 c.c. and 180 m.g. per 100 c.c., only four are below 140 m.g. per 100 c.c. and 14 are above 180 m.g. per 100 c.c. A correlation of the cholesterol findings to the degree and duration of symptoms was not possible. In the cases of ozena upon which I have operated, I was unable to establish a relationship between the postoperative result and the pre-operative cholesterol findings.

Summarizing, I cannot agree with Fleischmann's statement that low cholesterol content of the blood serum is found in atrophic rhinitis and ozena. In my examinations, with the elimination of all conceivable sources of error, the cholesterol level varies, keeping generally within normal ranges or slightly above normal, and shows no changes pathognomonic for either atrophic rhinitis or ozena.

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51 West 73rd Street.

THE VALUE OF HEMATOLOGY TO THE OTOLOGIST.*

DR. ARTHUR WEISS, New York.

In the short time allotted me, I shall not be able to more than skim over two routine laboratory procedures that I have found to be by far the most satisfactory in the diagnosis and prognosis of infectious disease. They are: *1*. The complete blood count with special stress on the morphological characteristics of the individual leukocyte; and *2*. the acceleration of erythrocytic sedimentation. The value and shortcomings of both of these tests in the diagnosis of existing inflammations has been elaborated by many. As long as no special attention was given to the nuclear structure of the neutrophil, the increase in the total number of leukocytes due mainly to a neutrophilia, and a progressive decrease in erythrocytes and hemoglobin, were the only indicators of the severity of any infection. With the introduction of the staff or immature neutrophil a definite aid to the clinician was uncovered. This emblem of the combat phase of infection was found to far excel the temperature curve or even the apparent clinical status of the patient, as a guide in prognosis and treatment. As a valuable adjunct to this, the sedimentation test has made it possible to follow the course of an illness, even after the blood picture has ceased reflecting any further abnormal findings. Its value is also inestimable in those chronic or localized conditions which for some unknown reason do not incite any leukopoetic changes.

Because of the narrow bony confines of the operative field, and its close proximity to those vital structures, the brain and spinal cord, any information that tends to facilitate diagnosis or detect lurking complications is certain of an enthusiastic welcome from the otologist. I shall not enter upon any discussion of the theories that attempt to explain the appearance of an increased percentage of staff cells. Nor shall I make any effort to definitely place the cause of accelerated erythrocytic sedimentation. Suffice to say, that the former is an evidence of stimulated bone marrow activity, while the latter is the result of absorption into the blood plasma of the products of inflammation. Inasmuch as both of these tests are non-

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specific biological phenomena, the findings are always proportionate to the amount of irritation and not the type of causative agent.

I shall now flash upon the screen a number of slides illustrating the characteristics of the staff cell, with some tables showing its activity in some otological conditions. I shall also demonstrate a sedimentation cylinder and the application of this test in ear infections.

Otitis Media Group: Table I shows case of otitis media resulting from a severe respiratory infection. It shows a gradual decrease in erythrocytes and hemoglobin with a marked leukocytosis and a high per centage of staff cells. Because of the persistence of these cells, patient was operated. After operation staffs return to normal.

TABLE I—OBSERVATIONS ON THE BLOOD IN CASE 1.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Lymphocytes	Eosinophils	Basophils	Monocytes	Comment
12/5	4.06	72	19,000	....	27	62	8	....	....	3	Macrocytosis, increased platelets
6	4.18	73	24,000	2	27	50	17	....	....	4	Toxic degeneration of neutrophils
7	4.16	73	22,400	1	21	55	20	....	....	3	Toxic degeneration of neutrophils
9	4.22	74	28,000	2	34	36	22	....	....	6	Toxic degeneration of neutrophils
10	4.20	74	26,400	4	34	40	17	....	....	5	Toxic degeneration of neutrophils
11	4.15	73	26,300	2	33	37	23	2	....	2	Toxic degeneration of neutrop., 1 myelocyte
12	4.03	72	23,800	2	32	41	19	....	....	6	Toxic degeneration of neutrophils
13	4.06	73	19,600	2	27	43	25	....	....	3	Toxic degeneration of neutrophils
14	4.16	74	19,000	2	20	39	32	1	....	6	
16	4.05	72	16,300	....	11	49	33	....	1	6	
17	4.41	69	24,300	....	11	71	12	1	....	5	
18	4.17	72	25,600	1	5	77	12	....	....	5	Poikilocytosis
19	3.99	70	22,300	....	5	80	10	....	....	5	
20	3.60	70	18,800	....	4	76	15	....	....	5	
21	3.46	69	13,000	....	5	73	18	1	1	2	
23	3.54	67	12,600	....	5	67	24	....	....	4	
24	3.41	65	12,600	....	5	68	20	1	....	6	

Table II. Another case of otitis media. This does not give any history of a preceding upper respiratory infection. In spite of an increase in leukocytes, the percentage of staffs is normal. No operation was deemed necessary and the patient was discharged with the condition improved.

TABLE II—OBSERVATIONS ON THE BLOOD IN CASE 2.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Staff Cells	Segmented Cells	Eosinophils	Lymphocytes	Monocytes
5/ 9	4.80	74	12,200	2	25	....	60	13
10	4.12	70	12,800	2	24	1	61	12
12	4.09	70	10,200	4	36	2	48	10
13	3.96	70	13,800	3	32	3	54	8
14	3.87	68	7,000	2	26	....	65	7

Mastoid Group: Table III: Acute mastoiditis with history of purulent discharge from ear for one week, which tended to increase in amount. X-ray showed destruction in the mastoid. Blood picture shows leukocytosis with slight increase in the percentage of staff cells. There is an inversion of the neutrophil-lymphocyte ratio.

TABLE III—OBSERVATIONS ON THE BLOOD IN CASE 3.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Staff Cells	Segmented Cells	Eosinophils	Basophils	Lymphocytes	Monocytes	Comment
2/10	4.18	66	11,800	7	59	1	....	29	4	
11	4.19	68	9,800	7	53	....	1	34	5	
13	4.17	67	13,000	7	57	1	1	30	4	
14	4.19	69	14,200	6	61	....	....	29	4	
15	4.20	67	13,400	6	72	....	....	19	3	
17	4.16	68	11,800	9	54	....	....	32	6	
18	4.10	69	11,000	9	66	....	....	20	5	Platelets increased
19	4.08	68	10,800	7	58	1	1	25	7	1 myelocyte, macrocytosis
20	4.04	67	20,800	7	74	....	....	16	3	Macrocytosis
21	4.00	66	28,800	10	75	....	....	13	2	Slight anisocytosis
22	3.89	66	20,600	7	74	....	....	16	3	Slight macrocytosis
24	4.20	68	10,000	7	61	....	....	27	5	Slight macrocytosis
25	4.16	69	11,600	5	67	....	....	23	5	Slight macrocystosis
26	4.05	68	9,600	5	65	....	....	24	6	Slight macrocytosis
27	3.87	68	11,800	5	65	....	....	22	8	Anisocytosis
28	3.64	67	9,600	4	64	....	....	25	7	Anisocytosis
3/ 1	3.89	68	11,600	4	68	....	....	21	7	

Chronic Mastoiditis: Table IV: Patient had a chronic condition of the ear for the past thirty years, off and on. For the past year aural discharge was constant. X-ray showed a destroyed mastoid. Blood series show little except for a leukocytosis. After operation the blood count gradually reached a true normal.

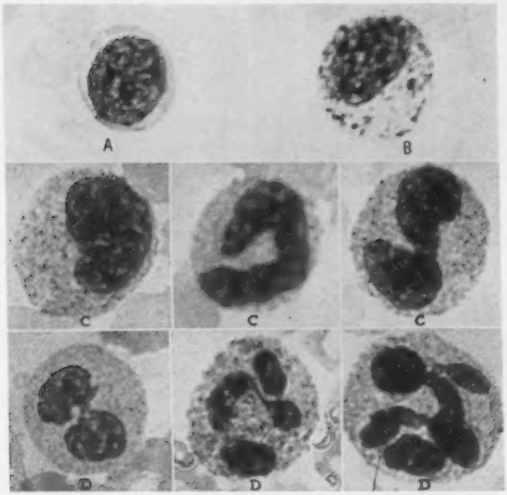


Fig. 1. Microphotographs showing the sequence of the granular system. (A) Myelobast, (B) metamyelocyte, (C) forms of staff or immature neutrophils as seen in blood films and (D) forms of mature or segmented neutrophils. As infections increase in severity the staff cells replace the mature or segmented neutrophil. The reverse occurs as the inflammation abates.

TABLE IV—OBSERVATIONS ON THE BLOOD IN CASE 4.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Staff Cells	Segmented Cells	Eosinophils	Lymphocytes	Monocytes
9/11	4.28	75	13,400	7	75	....	17	1
12	4.58	75	7,600	5	70	....	20	5
13	4.36	75	7,000	6	67	1	24	2
14	4.32	75	8,400	6	68	1	24	1
16	4.15	75	7,800	5	58	1	32	4
17	4.20	75	6,800	4	68	....	24	4
18	4.25	75	8,000	5	60	1	30	4
19	4.14	75	11,000	6	62	1	26	5
20	4.18	75	15,800	4	65	....	27	4
21	4.45	75	11,800	7	63	....	24	6
22	4.46	75	11,000	7	59	....	30	4
23	4.38	75	9,000	4	58	1	32	5
24	4.56	75	8,000	4	65	3	23	5
25	4.28	75	6,800	4	62	2	28	4
26	4.52	75	8,600	3	57	....	34	6
27	4.31	75	8,400	3	64	1	26	6
28	4.21	75	8,000	3	61	....	32	4
29	4.22	75	10,200	3	57	1	34	5
30	4.36	75	11,000	2	60	1	33	4
10/1	4.40	75	8,600	2	61	1	33	3

Recurrent Mastoiditis: Table V: Patient starts complaining of headache one week after discharge for apparently successful mastoid operation. Two days before readmission, ear and postauricular wound start discharging pus. During the first part of his stay he apparently improved, although the blood picture was not normal. With the return of his headache, temperature and right facial paralysis, it was deemed necessary to do a revision. Shortly thereafter the patient became irrational, comatose and died. Blood picture gradually became worse. Before death both the leukocytes and staff cells had reached very high levels. This case revealed a streptococcus mucosus on blood culture and culture of the spinal fluid.

TABLE V—OBSERVATIONS ON THE BLOOD IN CASE 5.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Eosinophils	Basophils	Lymphocytes	Monocytes	Comment
11/11	4.96	90	10,000	....	7	70	....	....	20	3	
12	5.07	90	7,200	....	9	53	....	....	36	2	
13	5.10	91	9,800	....	10	51	....	....	38	1	
14	4.89	88	11,600	....	9	65	1	....	22	1	
15	4.72	88	8,000	....	8	63	2	....	26	1	
16	4.82	89	13,000	....	10	50	1	1	36	2	
18	4.87	89	11,400	1	12	62	....	....	20	5	
19	5.05	86	13,800	....	10	72	1	....	14	3	
20	4.84	87	12,400	....	15	68	1	....	14	2	
21	4.73	88	15,000	1	15	66	1	....	14	3	
22	4.69	86	13,400	1	14	70	1	....	11	3	
23	4.70	86	12,000	1	10	69	....	....	17	3	
25	4.49	85	12,400	1	18	57	1	1	20	3	
26	4.78	84	12,200	2	15	68	....	....	11	4	
27	4.67	82	23,800	2	19	73	....	....	4	2	
29	4.58	78	34,000	3	32	54	....	....	7	4	Toxic degeneration of
30	4.38	76	32,600	....	33	62	....	....	4	1	neutrophils

Mastoids with Venous Complications: Table VI: Sinus thrombosis—Patient had been discharged after an apparently successful mastoidectomy. After being home a short while, started to run fever. On admission she had tenderness along the jugular vein. Blood picture showed a marked leukocytosis with a high staff count. In spite of ligated jugular and occluded lateral sinus, blood picture remained unchanged. After revision the blood picture rapidly returned to normal.

TABLE VI—OBSERVATIONS ON THE BLOOD IN CASE 6.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Eosinophils	Lymphocytes	Monocytes	Comment
7/12	4.80	60	14,400	....	26	69	....	4	1	
12	4.98	63	6,500	....	17	61	....	20	2	
13	4.72	65	12,700	....	20	58	1	19	2	Jugular ligation, occlusion of lateral sinus
14	4.56	65	10,100	2	34	39	....	19	6	
15	4.44	73	11,000	1	25	57	....	17	....	
16	3.68	60	16,000	1	19	52	....	28	....	Revision of mastoid
18	4.48	73	9,500	1	14	50	....	25	10	
19	4.24	70	8,500	....	4	36	2	57	1	
20	5.04	80	16,000	2	8	60	1	27	2	
22	4.84	78	8,700	....	9	50	....	38	3	
23	5.28	80	7,500	....	7	45	....	44	4	
26	5.84	82	16,000	....	3	41	....	51	5	
28	.....	....	.....	....	3	31	1	60	5	
30	4.32	75	9,300	....	6	42	....	46	6	
8/ 5	4.24	72	8,500	....	1	39	....	59	1	
9	4.08	75	7,000	....	2	34	4	59	1	

Primary Bulb Thrombosis: Table VII: Upon admission gave history of having had earache, aural discharge and fever. Paracentesis had caused relief, but with return of symptoms had been sent to the hospital. X-ray showed no mastoid involvement. Blood culture positive for streptococcus hemolyticus. Operated on same day. No mastoid pathology found. Sinus plugged at the knee. Blood picture during illness showed progressive anemia, very marked leukocytosis, with a high, and towards the end ever mounting, staff count. In spite of numerous transfusions patient succumbed.

TABLE VII—OBSERVATIONS ON THE BLOOD IN CASE 7.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Eosinophils	Basophils	Lymphocytes	Monocytes	Comment
11/9	4.610	75	23,400	2	19	68	....	....	8	3	
12	4.560	62	12,500	3	22	43	....	....	30	2	
13	4.180	62	11,400	1	18	53	....	....	26	2	Poikilocytosis
14	3.890	60	15,000	2	24	44	....	....	27	1	2 myelocytes
15	3.810	60	17,200	1	25	39	....	....	34	1	
18	4.060	61	24,300	1	15	33	....	....	49	2	
19	4.030	64	23,200	1	16	50	....	....	31	2	Toxic degen. neutrophils
20	4.160	63	18,600	....	9	41	....	1	47	2	Toxic degen. neutrophils
21	4.170	62	12,400	....	9	42	1	1	45	2	Toxic degen. neutrophils
22	4.190	61	15,200	1	26	50	....	....	20	3	Toxic degen. neutrophils
23	3.890	63	25,800	1	40	27	....	....	30	2	Toxic degen. neutrophils
25	3.695	58	19,600	2	47	32	....	....	17	2	Toxic degen. neutrophils
26	3.238	47	23,400	3	42	33	....	....	18	4	Toxic degen. neutrophils
27	3.250	49	26,200	2	40	36	....	....	19	3	Toxic degen. neutrophils
29	3.307	53	22,800	1	40	34	....	....	23	2	Toxic degen. neutrophils

Sinus Thrombosis with Fatal Termination: Table VIII: This case is of interest because it again illustrates the treacherous type of infection caused by the streptococcus mucosus, showing little or no hematological response until after operation, when the pathology spreads very rapidly and the blood picture reveals the hopeless condition of the patient.

TABLE VIII—OBSERVATIONS ON THE BLOOD IN CASE 8.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Lymphocytes	Monocytes	Comment
12/24	4.76	90	10,800	....	6	68	18	8	
26	4.68	87	9,000	....	4	65	29	2	
1/10	4.35	84	22,000	2	12	79	4	3	
11	4.28	85	18,600	3	25	64	5	2	1 myelocyte

Mastoids with Extra-aural Complications: Table IX: Acute Glomerular Nephritis—Shortly after discharge from the hospital, where mastoid had been exenterated, patient developed temperature. Some postoperative complication was suspected. Blood picture showed a severe anemia, marked leukocytosis and high staff count. The appearance of hematuria helped locate the cause of the hematological stimulation. With the clearing up of the nephritis the blood picture gradually returned to a normal differential.

TABLE IX—OBSERVATIONS ON THE BLOOD IN CASE 9.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Eosinophils	Basophils	Lymphocytes	Monocytes	Comment
11/30	2.79	60	15,800	....	23	55	....	....	22	....	
12/ 2	2.78	51	16,200	....	28	58	....	....	12	2	Marked anisocytosis
3	3.00	49	12,000	1	35	46	....	....	14	4	Marked anisocytosis, increased number of platelets
4	3.01	49	13,300	4	32	47	1	....	12	4	Polychromasia
5	3.25	47	13,600	3	33	40	....	....	21	3	
6	3.47	47	15,300	1	24	50	1	....	20	2	2 myelocytes
7	3.37	47	10,000	1	20	46	1	1	28	3	
16	3.53	47	14,200	2	20	65	....	....	10	3	
24	3.56	50	13,000	....	7	63	2	....	24	4	
28	3.72	53	16,000	....	9	43	2	1	41	4	

Diabetes and Erysipelas: Table X: Patient had pain and purulent discharge from the ear for three weeks. X-ray showed mastoiditis.

Blood picture had been apparently normal until after the operation. With the development of erysipelas, the leukocytes rose rapidly and the staff cell count rose to 48 per cent, resulting in death of the patient.

TABLE X—OBSERVATIONS ON THE BLOOD IN CASE 10.

Date	Red Blood Cells	Hemoglobin	White Blood Cells	Mast Cells	Staff Cells	Segmented Cells	Eosinophils	Lymphocytes	Monocytes	Comment
3/16	4.00	75	8,800	....	6	69	1	24	....	
17	4.06	70	13,200	....	11	79	....	9	1	
19	4.16	70	22,000	....	8	82	....	10	....	
20	4.05	70	29,000	....	6	82	....	12	....	
21	3.90	70	20,600	....	41	49	....	9	....	1 myelocyte
22	4.51	75	18,600	2	48	34	....	15	....	1 plasma cell

After viewing a number of slides illustrating the blood changes occurring in a selected group of otological conditions, the following observations become evident:

1. The height of the staff count is not a satisfactory guide as to the urgency of operation in acute infections of the middle ear.

2. The leukocyte and staff counts are often higher in acute purulent otitis media than in acute mastoiditis.

3. The blood picture in acute and chronic mastoiditis show no outstanding picture. The recurrent mastoid, if caused by the streptococcus mucosus, will show definite changes.

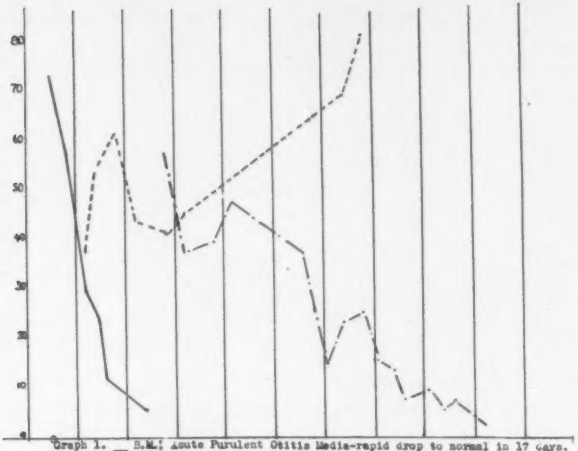
4. Cases of brain abscess or meningitis do not cause marked changes unless they are the result of a generalized infection.

5. All cases of mastoiditis with venous complications show marked changes in the blood picture.

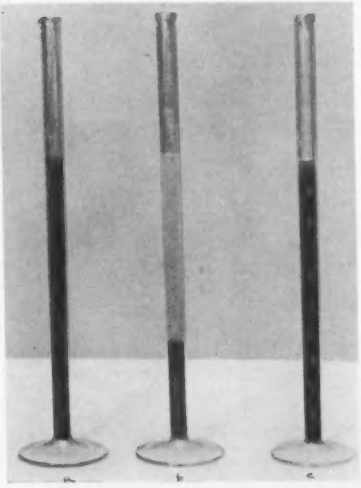
6. Any rise in the percentage of the staffs after operation indicate the presence of some complication.

7. The complications need not be in the operative area, but may involve some other portion of the body.

Before entering upon what information we can expect of the sedimentation test, allow me to show you a sedimentation cylinder that I have found to be an accurate and by far the most practical means of measuring acceleration in sedimentation. (Fig. 2 shows three cylinders: *a*. immediately after filling; *b*. pathological case, after



Graph 1. ——— S.M.; Acute Purulent Otitis Media-rapid drop to normal in 17 days. Patient discharged without operation.Graph 2. —— A.M., Diabetic, Strep. Mucosus O.M.P.A.-Mastoid. Sedimentation highest toward death. Graph 3. _ . _ . I.S.;Diabetic,Mastoiditis. Pt discharged although sedimentation was increased returned for revision after which sedimentation returned to normal.



45 minutes; and *c*. normal sedimentation after 45 minutes.) The cylinder is 200 m.m. long and is calibrated to 150 m.m. Calibrated portion holds 2 c.c. Graph I shows three curves (as per negative).

The sedimentation test employed in a large series of otological conditions yielded the following data:

1. All cases of O. M. P. C., chronic mastoiditis with cholesteatoma, or even labyrinthine symptoms, gave normal readings.

2. Acute furunculosis of the external canal gave normal readings.

3. O. M. P. A. and acute mastoiditis gave high readings (as high as 80 per cent).

4. Whereas the rate dropped rapidly in cases of O. M. P. A., requiring no operation, cases with mastoid involvement had rates that stayed high until after operation.

5. Sedimentation rate rises somewhat after operation, and drops very gradually (often four to six weeks).

6. When done in series it will detect postoperative complications. It can also be used as an indicator for the necessity of mastoidectomy in cases of O. M. P. A.

Before finishing this paper, I wish to stress two points that I may not have emphasized sufficiently. Inasmuch as both of these phenomena are nonspecific they cannot be used for diagnosis. However, if used in conjunction with one another and done in series (blood count daily, sedimentation test every four to five days) much will be gathered of definite help to the otologist.

Beth Israel Hospital.

THE MANAGEMENT OF THE BIOPSY IN LARYNGEAL CASES.*

DR. ALFRED PLAUT, New York.

The good management of the biopsy in laryngeal cases depends upon the clinician as well as upon the pathologist. Unless the right spot is chosen for excision and the right time, unless a sufficient amount of tissue is submitted, and proper fixation and correct labeling have been done, even the most experienced pathologist cannot give a satisfactory diagnosis. The indication for biopsy entirely depends upon the clinician. When the laryngologist is satisfied that a certain ulcerative lesion on a vocal cord is tuberculous in character, he certainly will not take a biopsy, and the carcinoma which may be hidden under the ulcer grows on beyond possibilities of cure. It must be stated that tuberculosis of the lungs and carcinoma of the larynx can occur in the same patient. The danger of taking a biopsy is very slight. In ulcerative lesions it is practically nonexistent. Even in closed lesions a biopsy should be done unless the diagnosis is absolutely established. An ordinary inflammation or a benign papilloma may be situated on top of a more deep-seated carcinoma.

A number of lantern slides are demonstrated, not with the idea of showing histological pictures of laryngeal cases but with the intention of emphasizing some of the pitfalls in laryngeal biopsy work.

Edematous connective tissue, for instance, is shown side by side with tissue from a true myxoma of the thigh. Myxomata are very rare tumors; they hardly ever occur in the larynx. A diagnosis of myxoma should not be accepted unless definitely proven by a competent man.

Very thick squamous epithelium, with or without hornification, in itself has nothing to do with malignant change. Poorly prepared sections through very thick squamous epithelium often have misled insufficiently trained people to a diagnosis of carcinoma where no carcinoma existed.

Several specimens are shown which mostly consist of normal laryngeal tissue or of benign polypoid and papillary structures. Small spots at the edges or in the center, however, consist of carcinoma.

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*Read before the New York Academy of Medicine, Section of Otolaryngology, March 16, 1932.

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These specimens show how a biopsy taken only a little less deep would have made it impossible for the pathologist to recognize the carcinoma.

Pachydermia is characterized not only by the thickening of the squamous epithelium but by the intense inflammation in the underlying connective tissue which probably precedes the epithelial thickening. The presence of many mitotic figures in pachydermia or in other epithelial thickenings in the larynx does not justify us in voicing a suspicion of malignant change. Any unusual change in the squamous epithelium in the larynx must first be analyzed to see whether or not it may be due to cauterization, radiation or any other form of treatment.

In very rare instances the biopsy removes the whole carcinoma. A slide is shown where a fragment hardly the size of a pinhead represents the only carcinomatous spot in a large amount of material. The same patient on two further biopsies, with removal of large amounts of tissue, did not show any carcinoma.

Our aim in the management of the biopsy is to shorten the time elapsing between the onset of hoarseness and the institution of cancer treatment. Only close co-operation between the laryngologists and the pathologists will lead to that goal.

Stuyvesant Park, East.

RECENT ADVANCES IN THE APPLICATION OF BIOCHEMISTRY TO OTOLOGY.*

DR. ELLA A. FISHBERG, New York.

The total contributions of biochemistry to otology up to the present time may be summed up in the short sentence, *"Multa non multum,"* but future work along the lines already started will certainly give results that will be of actual practical value to the otologist.

The problem of otosclerosis is one on which much work has been done, especially in regard to the interpretation of otosclerosis as a single manifestation of a general metabolic derangement, and we may distinguish three main lines along which these investigations have been carried out. It has been interpreted successively as a disturbance of uric acid, calcium and lipoid metabolism.

The view of otosclerosis as a single manifestation of a general metabolic condition is by no means a new one. In 1857, Toynbee advanced the theory that stapes ankylosis was a form of rheumatic gout of the stapediovestibular joint, and claimed that patients manifesting this condition usually showed a uric acid diathesis. On the basis of these findings it was pointed out that this form of stapes ankylosis was more common in women and that many women noted a distinct decrease in hearing after each pregnancy. Dickie, in 1903, concluded from a clinical study of otosclerosis that it was a uric acid toxemia due to the retention of this metabolic product which attacked the individual in his most vulnerable point, the labyrinth. With the development of the finer methods of blood analysis, it was shown that there was no constant increase of the uric acid in the blood and that the above theories were without foundation.

The evidence of a disturbance of calcium metabolism in otosclerosis started with the work of Mayer, who pointed out on the basis of his pathological studies of otosclerosis that the disease process was characterized by a proliferation of the fibrous marrow and might be designated as a fibrous ostitis resembling the condition described by Recklinghausen in the other bones of the skeleton. There were distinct cysts found in the otosclerotic foci in one of Mayer's cases. This pointed to an essential relationship between the two conditions.

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Later determinations of the calcium in 32 cases of definite otosclerosis were reported by Leicher. In 75 per cent of these cases, *i. e.*, 24, the blood calcium was found to be diminished below the level of normal variation. He claimed evidence of the disturbance of the calcium metabolism in six of the eight normal cases, since after administration of primary sodium phosphate for two weeks there was a definite lowering of the calcium content of the serum; while in 20 normal persons under the same experimental conditions no change, or at most a slight change, in the calcium content could be shown. Kopetzky and Almour found a definitely diminished blood calcium in cases of otosclerosis. Leicher was able to elicit positive Erb and Chvostek signs in these cases, due to the heightened neuromuscular irritability caused by the calcium diminution in the blood. It has been shown that in any normal person the injection of either neutral or basic phosphate solution will lower blood calcium and induce tetany. Acid phosphate solution will not produce tetany but does cause a decrease in blood calcium. Tisdall obtained exactly similar results. I therefore cannot see how the experiments of Leicher can in any way prove a special lability of the calcium metabolism in otosclerotic individuals. It is worthy of mention that most of the work done on the calcium content of the blood has not been characterized by sufficient precaution in obtaining really standard conditions. This fact is emphasized by Collip in his Harvey lecture. He points out that the very fact of keeping blood in a cool room for a few hours causes a distinct lowering of the serum calcium; in fact, to such an extent that a pathologically lowered calcium value is simulated.

It should be mentioned in passing that the many cases of otosclerosis in combination with the condition of fragilitas ossium and blue sclerae seem to point to some common calcium derangement.

The experimental work on the connection between the parathyroid gland and calcium metabolism culminated in the isolation by Collip of a physiologically potent parathyroid hormone. Extensive investigations have been made on the relation of this hormone to calcium metabolism in general and to otosclerosis in particular. The injection of parathyroid extract Collip led to decalcification of the skeleton. Jaffe and Bodansky showed that in the regions of greatest decalcification changes appeared, resulting in the formation of the typical lesions of fibrous ostitis. Gale and Jaffe then investigated the condition of the labyrinth during acute and chronic hyperparathyroidism. They showed that the labyrinthine capsules of these animals were characterized by a complete absence of change, although the

adjacent skull bone (dependent on the age of the guinea pig, duration of treatment and dosage), showed various degrees of resorption, with evidence of osteoclasts and fibrous tissue. The capsule, even in closest proximity to the skull bone, showed no such changes. The bony capsule of the guinea pig did not show resorption after treatment with parathyroid extract because this bone had a lower rate of metabolism and its mineral salts were less readily available, due to the fact that it was a region of inactive bone growth. Whether this work can be applied to other animals remains as a question for future workers and certainly deserves attention along the lines worked on by Jaffe.

Aub has shown that there is a reserve supply of calcium available to protect the vital structure of the bone. Some special parts may store calcium in large amounts. One patient on large doses of calcium chlorid stored 14 grams of calcium in six days. This deposition took place mainly in the spicules of bone which ramify through the bone marrow. In rabbits the ingestion of parathormone produced a distinct loss of trabeculae as shown in X-ray and postmortem examination. With this reserve calcium storage mechanism it was obvious that the removal of actual calcium deposits from any particular tissue would be extremely difficult. Aub proposed the use of the otosclerotic ear as a measure of the efficacy of calcium removal, because the hearing would presumably furnish a delicate indicator for the amount of calcium taken out. He induced the elimination of many grams of calcium, but very little of this could be proven as having come from the ear. An experimental study was then undertaken by Aub and Crockett as to the possible curative effect of parathormone in otosclerosis. This has now been going on for some years. In a personal communication from Aub as to the progress of this work, he states: "We are watching these cases over a long period of time to see the ultimate results. I think it fair to say that some of the early cases of otosclerosis have shown improvement on a low calcium diet with parathormone or ammonium chlorid added in order to try to decalcify. The more advanced cases have shown no results at all and I am not sure how long the improvement of the early cases will last. On the whole, the results have not been startling. I think the reason for this is the excellent reserve supply of calcium in the trabeculae of the bone."

The possibility of lipoid metabolism disturbance in otosclerosis was emphasized by Brunner from a case of Gaucher's disease, deaf during life, in which pathological examination showed typical otosclerotic foci in the petrous bone, with characteristic Gaucher's cells in

some portion of these foci. Like otosclerosis, Gaucher's disease is familial and occurs predominantly in the female sex. Berberich, working in the Frankfurt Otological Clinic, reports extreme decreases in the cholesterol content of the blood of persons manifesting otosclerosis. He reports values as low as one-tenth the normal figures, *i. e.*, as low as 20 m.gm. per cent. I have never seen blood cholesterol values anywhere within the range of those reported, 90 m.gm. per cent being the lowest ever encountered by me in a general hospital service. Since similar values were reported in cases of ozena from the same clinic, which, on being checked in my laboratory by Dr. Wachsberger, gave absolutely normal figures, the impression remains that the cholesterol figures as reported from this laboratory must be regarded with a certain measure of skepticism.

Otosclerosis has also been regarded as a dietary and vitamine deficiency in analogy to rickets. Reports of the diminution of blood phosphorus in otosclerotic patients have not been lacking. Feeding experiments by Kauffmann with diet deficient in vitamins resulted in rachitic changes in the bone, with osteoid changes in the capsule of the labyrinth, the cochlear and the stapediovestibular region. They claimed that healing processes in the temporal bone similar to those following rickets in the long bones gave a picture identical with that of otosclerosis. Hence, otosclerosis may be a result of rickets or a dietary deficiency similar to that which causes rickets, still extant during adult life.

In an attempt to arrive at some sign of bone involvement in suppurative ear disease, Friesner and Rosen analyzed the pus from various sources and also that from discharging ears. They compared the calcium content of pus from the ear with frank bone necrosis with the calcium content of discharging ears without demonstrable bone necrosis, and from abscesses elsewhere in the body. While the calcium content of cases of liver abscess, empyema, acute purulent otitis media, showed a calcium content not above 7 m.gm. per 100 c.c., the cases of acute mastoiditis had values ranging around 30 m.gm. per 100 c.c., with extremes of 20 to 48 m.gm.

Passing to the chemistry of the spinal fluid in otological conditions, we come to more objective work. It was first pointed out by Kopetzky that the spinal fluid in purulent meningitis was distinctly less alkaline than normal. This fact the author brought into correlation with the tendency of the brain tissue to absorb water and thus cause the pressure symptoms noted in meningitis. On repeating these experiments we have noticed a distinct lowering of the pH of the spinal fluid during the progress of meningitis. Kopetzky in this early piece of

work showed a large decrease in the amount of reducing substance in spinal fluid. This fact provides a means for very early diagnosis of meningeal involvement. Other workers have brought this fact into correlation with the amount of sugar in the blood and have obtained ratios between the blood and spinal fluid sugar which are helpful in the differential diagnosis of the various forms of brain involvement following ear infection. The normal sugar of cerebrospinal fluid as measured by the Folin-Wu method is 40 to 65 per cent. This relationship is considered fairly constant by most workers and does not change in diabetes or after insulin injection because the changes of the spinal fluid sugar and blood sugar run parallel. In cases of tuberculous meningitis, where the blood sugar is high, even the normal spinal fluid value will give a low ratio. In purulent meningitis this ratio falls to from 0 to 35 per cent, while in epidemic polyomyelitis, brain tumor, epidemic encephalitis, it is normal or usually increased. In purulent spinal fluids Kopetzky found that the ratio of sodium to potassium was shifted in favor of the potassium. In purulent meningitis the chlorids are low (from 600 to 650 m.gm. per 100 c.c.), in contrast to the normal 675 to 750 m.gm. per 100 c.c. A further study of this spinal fluid, blood-chlorid gradient may prove of value.

Summing up, we may say that definite facts for the use of the practicing otologist, except in the chemistry of the spinal fluid, have been few, but that further work along the lines of general calcium and lipoid metabolism are the most promising fields for future work.

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THE PROGNOSTIC VALUE OF STREPTOCOCCIC SUBCULTURE IN AFFECTIONS OF THE EAR.*

DR. L. G. HADJOUPOULOS, New York.

The hemolytic streptococcus is certainly the most prevalent of all organisms encountered in the study of the bacteriology of mastoid infections. In the group of pyogenic micro-organisms the types commonly designated as streptococci are the least homogeneous in their biological and clinical characteristics. In relation to mastoid pathology the finding of hemolytic streptococci in a bacteriological examination is of no more significance than a report of any other pyogenic micro-organism, be it staphylococcus, pneumococccus or even meningococcus.

Clinical pathologists differentiate at least four types of mastoid infection: *1*. the acute hemorrhagic-necrotic, *2*. the acute purulent-coalescent, *3*. the subacute and chronic of the above types with their recurrent forms, *4*. mixed types, both necrotic-coalescent. Even this clinical classification is not of much value for the prognosis of the subsequent course of the disease. From the operative findings alone it is impossible to prognosticate the subsequent course of the disease because the same complications and sequelae may occur in any of the pathological forms encountered. A careful biological study of streptococci encountered in otitic infections has been valuable in this respect.

Classification of streptococci according to their hemolytic properties is of little value because in a great majority of acute pyogenic infections the hemolytic strains are the most predominant. A classification of streptococci based on their metabolic functions in deriving energy from the fermentation of certain sugars is found to be of greater service.

The choice of lactose, salicin and mannite according to the method of Holman combines certain unique features. It limits the number of sugars to three, and therefore results in only eight possibilities; while with the use of 10 sugars as advocated by some investigators, the number of possibilities rises to over a thousand. It eliminates

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duplication since it does not use sugars that are chemically similar in structure. Of the three "sugars" mentioned above, lactose is the only one that may be regarded as a sugar in the true chemical sense. Salicin is a glucoside containing the sugar in combination with the benzene ring, while mannite (manitol) is really a dibasic alcohol.

The ability to ferment simple sugars is characteristic of a great majority of streptococci. The fermentation of a mono- or disaccharid requires a number of oxygen atoms equivalent to the carbon present in the particular sugar. Since oxygen is freely accessible on open surfaces, streptococci that depend solely on carbohydrates for their energy requirements are found mainly on such surfaces, as in the case of the mucous membrane of the upper respiratory and alimentary tracts. These streptococci are essentially obligate aerobes and very prevalent in otitic infections. Once they become aggressive they follow the course of the nasopharyngeal mucous membrane, invade the middle ear through the Eustachian tube, and then through the antrum into the mastoid cavity. They give rise to a pathological picture characterized by an acute catarrhal hemorrhagic and even necrotic inflammation. They cannot penetrate deep into adjacent tissues owing to the lack of oxygen prevailing there. Consequently the complications are not far-reaching. We may have a cortical perforation or sinus wall granulation, dural and sinus plate necrosis, and not very uncommonly a transient septicemia. The mortality is very low; in fact, we have actually had no deaths in our series of over 50 cases. This comprises the first group, *i. e.*, the lactose fermentation group of which streptococcus angiosus is the prototype.

In the second group to be discussed we have in addition to lactose fermentation, the salicin fermenters. The fermentation of salicin, a glucoside, containing a hexose base in a benzine ring, requires the splitting of a closed ring. The group of streptococci capable of performing this function do not depend on the free supply of oxygen, as was seen to be the case in the former class. Actually, they can function under abnormally reduced oxygen tensions. They, therefore, can invade deeper structures and give rise to a chain of grave complications that may eventually prove fatal. The pathological picture is that of an acute suppurative inflammation similar to the acute suppurative coalescent mastoiditis as differentiated by the clinician. An area much wider than the middle ear and mastoid process may be involved and may give rise to subperiosteal abscesses, sinus plate perforation, sinus phlebitis, sinus and jugular thrombosis, meningitis, cerebellar and cerebral abscesses, septicemias and, finally, remote

metastatic abscesses in muscle sheaths and bone marrow. We may, therefore, expect a higher percentage of mortality in this group, whose prototype is streptococcus pyogenes. In 58 cases of this category we had 20 deaths, a mortality of 35 per cent.

The third major group comprises those streptococci that are characterized by an unusual adaptability to focal environment. This may partly be explained by their ability to ferment not only carbohydrates and glucosides, like lactose and salicin, but also the by-products of previous fermentation by saprophytic as well as pathogenic organisms. Therefore, they can thrive in any focus whatsoever, although their selective habitat is in closed sacs, as, for example, in a tooth root abscess, and especially in the intestinal tract. Pathologically, they give rise to chronic granulating wounds in the mastoid process or elsewhere. Irrespective of their far-reaching fermentative properties, they give rise to fewer and relatively simpler complications as compared with the previous class . The mortality is very low. The outstanding feature of this group of streptococci is their predisposition for causing chronic lesions.

The three above mentioned classes do not complete the streptococcic bacteriology of mastoid infections; cyclically, we may find some of the rare groups predominating in certain years, as was the case last year with subacidus. Having not yet completed our studies of this group, I cannot enter into any discussion of their characteristics. Similarly, I have omitted the group of pneumococci and viridans streptococci in presenting the etiology of otitic infections. I should mention, however, that among pneumococci, the Group III pneumococcus mucosus capsulatus is the pneumococcus of considerable significance that gives rise to grave complications and causes a relatively high mortality.

Beth Israel Hospital.

THE USE OF "NEGOCOLL" AND "HOMINIT" IN THE MAKING OF MOULAGES AS AN AID TO PLASTIC SURGERY.*†

DR. SAMUEL IGLAUER, Cincinnati.

For many years there has been a need for some material with which impressions of various parts of the body could be taken with ease, certainty and comfort to both the patient and the physician. After many experiments, the late Dr. Poller,[1, 2] of Vienna, succeeded in producing such a plastic substance, to which he gave the name of "Negocoll." Negocoll is an elastic hydrocolloid which forms a thick paste upon being heated and which, on cooling and returning to the gel state, has a soft rubber-like consistency.

Uses: With Negocoll it is comparatively easy to obtain three dimension moulages which are valuable for study and planning prior to plastic operations (see Fig. 1), and which afford a permanent record of the deformities (see Figs. 2, 3, 4 and 5). Negocoll is also useful for the permanent reproduction of gross anatomical or pathological specimens. It may be employed in medicolegal cases to record wounds or injuries, or for the identification of criminals. Reproductions of rare artistic or archaeological objects can also be made for distribution to collectors or museums.

Technique: To take an impression of any part of the body the Negocoll is heated and stirred in a double boiler until it liquefies, when it is allowed to cool to approximately body temperature. It is then applied directly with a brush, spatula or syringe in successive layers until a thickness of about one inch is attained. The inclusion of air bubbles should be avoided. As soon as it cools and solidifies (and this may be hastened with compressed air) it is lifted as a complete negative. Since it is nonirritating, it may be applied directly to the most sensitive parts—even the cocainized eyeball—and, owing to its elasticity, it may readily be withdrawn intact from under cuts or irregular surfaces.

In taking a cast of the face, breathing need not be interfered with, since the Negocoll may be applied within the vestibule of the nose without completely occluding the airway.

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Fig. 1. Patient holding a moulage of her face for comparison.

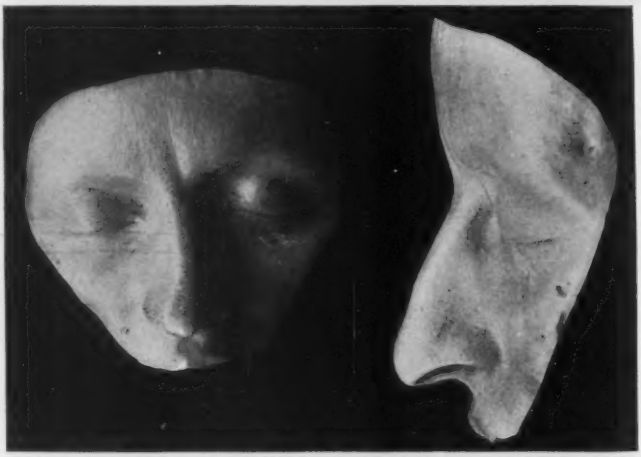

Fig. 2. Moulage of a patient with elongated nose and a furrowed brow.

To make a complete life mask of the head it is necessary to remove the plastic material in sections. This is accomplished by placing long strings in contact with the head prior to the application of the Negocoll. Upon pulling on the ends of the strings the negative is cut through and comes away in sections, which can then be assembled. In making a mask of this size it is advisable to reinforce the Negocoll by inserting wires between the various layers as they

Fig. 3. Same patient as Fig. 2. after shortening of the nose and removal of the furrow.

Fig. 4. Moulage of an aviator with a broken nose.
Fig. 5. Moulage of the same patient after the implantation of a piece of rib cartilage.

are being brushed on, or the Negocoll can be covered with a thin layer of plaster of Paris before the draw is made.

Owing to its resiliency it is possible to obtain a complete negative of the hand over which the composition has been applied like a glove,

from which the subject can withdraw his hand, leaving an intact negative.

Since Negocoll can be sterilized by boiling, it may be applied directly to fresh wounds in order to demonstrate various stages of an operation (see Fig. 6).

The Positive: For the production of the positive Poller prepared another compound, to which he gave the name "Hominit". This is a firm, resinous substance which is provided in various colors. Skin-colored Hominit is labeled "Carna".

Hominit melts at a temperature between 150° and 200° Centigrade, and is heated in a metal container. (The operator should protect his hands with heavy cotton gloves.) Using a heat-resisting brush, it is

Fig. 6. Moulage of a patient showing flap brought down from forehead illustrating one stage of a rhinoplasty.

then brushed or poured into the Negocoll mold, previously prepared, as described above. Care must be taken to avoid the inclusion of air bubbles and to prevent the formation of steam, which may occur if the negative is too moist. While the Hominit mass is still hot, strips of gauze are incorporated in it for reinforcement. For permanent specimens the Hominit cast should receive further reinforcement with a third Poller compound, "Celerit." If the latter is not available, plaster of Paris may be used instead. The cast is now withdrawn from the mold and the two separate very easily. The positive may be retouched if necessary. Any irregular projections can be removed with a knife, file or sandpaper, and any abnormal depression can be filled with a few drops of hot Hominit, using a

heated spatula. If desired, the moulage may be tinted with water or oil colors applied lightly with a brush.

Advantages: The Negocoll- Hominit technique has certain distinct advantages over plaster of Paris, which hitherto has been the chief substance used in making casts. Negocoll does not set rapidly and can be applied slowly and deliberately. It does not flow freely and, therefore, can be used on the face with the patient in the sitting posture. It is much lighter than plaster and, consequently, does not distort the features or push the eyeballs into their sockets. It does not adhere to or pull out the hair. In making moulages of the face no breathing tubes are required. Unlike plaster, it may be applied to undercuts and subsequently be removed intact without injuring the mold. Finally, the impressions taken with Negocoll give much finer details than those obtained with plaster.

The initial cost of Negocoll is much greater than that of plaster, but it can be used over and over again without deterioration.

In conclusion, I wish to express my appreciation to Morris Gruenebaum, D.D.S., who first called my attention to these plastic materials, and who, with Dr. Henry Gruener, has been of great aid to me in the production of some of the moulages presented today.

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707 Race Street.

A NEW METHOD OF TREATING SUPPURATIVE DISEASE OF THE NASAL SINUSES BY THE USE OF WARM MEDICATED VAPOR CONTAINING NASCENT IODIN.

Dr. Walter A. Wells, Washington, D. C.

It has happened too much recently that the tried and true old-fashioned remedies have been undeservedly displaced and pushed into the background of our therapeutic armamentarium.

The reason for this is in order that they make room for the latest products of big pharmaceutical houses who leave no stone unturned in their aggressive propaganda to impress the medical profession—a propaganda moreover which too often insidiously trickles out among the laymen, and may reach, sooner or later, such a proportion that we are made to feel that we are not up-to-date unless we sanction their latest therapeutic fad.

Iodin is indeed an old remedy, and our forefathers often used it with a success that fails in applying some of its much lauded substitutes. On empirical grounds alone it was employed in many bacterial infections. Modern scientific study has explained its efficacy.

It is no doubt one of the most valuable bactericidal agents which we possess. While mercuric chlorid in dilution of 1 to 1000 requires a thirty-minute exposure to render sterile a culture of streptococcus pyogenes, the same effect is reached in two minutes with a 1 to 500 dilution of iodin.

Very recently, Miller and Appleton (*Dental Cosmos*, 1931, p. 74) made laboratory studies in the sterilization of the oral field for operative purposes.

In comparison made with a number of new highly vaunted modern antiseptics, they came to the conclusion that iodin was of all the most efficacious. They discard the tincture of iodin U.S.P. as far too concentrated and therefore irritating to the membrane — and employ a solution of not higher than 2 per cent.

Lawall and Tice, in a study of iodin solutions as antiseptics (*Jour. Amer. Pharmaceutical Assn.*, 1932, XX, No. 2), showed that for wound irrigation a solution as dilute as 1 to 1000, was of surprising effectiveness from a bacteriological standpoint. Five c.c. of this solution were found to kill 1 c.c. of 24-hour culture of B. typhosus

and also of staphylococcus aureus within two minutes. Five c.c. of 1-10 dilution killed 0.1 c.c. of a 24-hour culture of B. typhosus within two minutes.

In the light of such experiments we well may accept the claim that is made that two drops of tincture of iodin added to large glass of water under suspicion will guarantee its safety for drinking purposes.

One of the most important discoveries that has come out of the study of this drug is that its therapeutic value is tremendously increased if the molecules can be brought into contact with the diseased tissue in their nascent state.

Indeed, it is a question whether or not the efficacy of iodin, whenever and in whatever form used, may not be ultimately ascribed to the presence of a certain amount of the agent in nascent form.

Even when applied locally to the skin, in the ordinary preparations used, some iodin is volatilized and, passing through the superficial epithelium, is absorbed and enters in the lymphatics. The good effect of iodin locally is limited to the fact that the unaltered molecule coagulates the albumin of the tissues and acts as an irritant. The nascent molecule acts without irritation.

It is well known that such valuable antiseptics as iodoform, aristol, iodol and nosophen depend essentially upon their peculiar property of releasing iodin in its nascent form.

Another factor very important for the beneficial effect of iodin is that the tissues or surfaces which we aim to influence should be as dry as it is possible to make them.

Quite recently there has come into use a method of treating chronic suppurative disease of the middle ear, by which very remarkable success is being obtained for conditions which have proved resistant to all previous methods of treatment.

The preparation used consists of an intimate association of iodin with boric acid, prepared by a special pharmaceutical process.

Without doubt the virtue of this remedy is due to the property it possesses of slowly giving up nascent iodin when brought into contact with the tissues of the middle ear.

Dr. M. D. Lederman (THE LARYNGOSCOPE, St. Louis, June, 1930), to whom we are indebted for its introduction into otological practice, lays great stress upon the importance of having a dry field.

He discards irrigation of the ear, specifying that the necessary cleansing must be accomplished by careful and thorough swabbing and gentle suction.

The success of the Lederman method in middle ear suppuration was my inspiration to attempt something similar in the suppurative disease of the sinuses.

It has been recommended that this dusting powder be insufflated also into the sinuses and a special apparatus has been designed for the purpose. But it is obvious that one cannot provide a dry field in the sinus cavities in the same manner as in the tympanic cavity and a dry field is an important condition for the success of the treatment.

It occurred to me that this might, however, be achieved by superheated air, which on account of its hygroscopic properties would have marked drying effect upon membranous lining of the sinuses, and that this air might at the same time be utilized for volatilizing the iodin, and delivering the drug in a nascent state into the interior of the cavities.

The preparation we use is in the form of a powder, containing iodin, boric acid, camphor, menthol and powdered cubeb, each 4 per cent; thymol, 1 per cent. We found that the addition of the cubeb was effective in neutralizing the pungent and irritating properties of the iodin.

The apparatus for volatilizing the mixture is a cylindrical-shaped hollow electric heater of bakelite composition, one end of which is closed with the exception of small central tube to admit a current of air, while at the other end is a removable cap, in the center of which is a tube for the exit of the air.

In order to leave hands free for necessary manipulation the heater is suspended from the top of a flexible rod fixed upon a stand, which makes it possible to place the apparatus at any desired position with reference to the patient.

Air is provided by a small rubber tube, passing from a compressed air tank to the inlet of electric heater, whereby we obtain a steady stream of air which can be regulated at will.

The real problem was how to deliver the volatilized gas into the interior of the sinuses.

For this purpose, special cannulae were devised individually for the frontal, maxillary and sphenoidal sinuses. The ethmoidal, by reason of its complex structure, does not lend itself in a satisfactory way to any method of interior medication.

The cannulae are all constructed with their proximal ends (or mouths) of the proper dimension for the small rubber adapter, such as is ordinarily used for the Eustachian catheters—this for the purpose of insuring the best connection with the soft rubber tubing.

We can utilize as a passageway for the heated air and medicated fumes either the natural openings of the sinuses, or we can make artificial openings expressly for the purpose.

The natural opening is available without preliminary operation in the case of the maxillary sinus, in about 60 per cent of the patients; in the frontal about 20 per cent, and the sphenoidal about 10 per cent.

The maxillary sinus or antrum, the one most frequently affected, is fortunately the one best adapted to fumigation treatment.

Those cases in which the cavity is not accessible through the natural openings, it is an easy matter to render it so by a puncture at the location of the inferior meatus.

We have devised a special trocar and cannula, very convenient for the purpose. The needle is separate from the handle, and latter can be set at different angles—a great advantage at times, especially in attacking the sinus of the right side. The mouth of the cannula is hollowed to the correct size for the conventional hard rubber adapter.

If we intend to repeat the treatment a number of times, then it is advisable to make a large artificial opening in the nasoantral wall of the inferior meatus—the so-called window resection.

This opening will remain permanent if made sufficiently ample and properly done. One may use for the purpose, the combined antrum trocar, devised a number of years ago by the author, or such other methods as may be preferred. In the case of low lying inferior turbinal, partial resection is advisable.

Many patients, when they apply to us for treatment have already had the window operation performed, for apparently there are certain specialists who harbor the delusion that this procedure is in itself sufficient to bring about a cure.

But drainage alone is not sufficient to cure a genuinely chronic case, and besides we must not suppose that the whole drainage problem of the antrum is solved because a free opening is made at its most dependent situation.

In the first place, it will not be at the most dependent situation (and therefore not the most favorable for drainage by gravity) when the individual is reclining, which is about one-third of his time, and in the second place, the movement of the cilia, Nature's own method of draining, is constantly upward, and therefore away from the artificial opening.

The chief advantage of a large artificial opening is, according to my idea, that it affords a convenient avenue through which to direct subsequent treatment.

The frontal sinus, as stated, can be naturally probed in about one case in every five. That is because, in about this proportion of cases, we find that the infundibulum, instead of merging into the meatus, opens directly upon the lateral wall just in front of this situation.

In all other cases it is necessary to remove the anterior end of the middle turbinal, close to its line of attachment.

The sphenoidal sinus is not naturally accessible to the probe or cannula in more than one case out of ten. It can, however, always be rendered so by taking off a slice of the middle turbinal in its most bulging part.

As it happens in the cases where these minor operations need to be done for the purpose of admitting the cannula, they are indicated besides by the diseased condition present.

It is now something over a year since we began to experiment with the use of iodin fumigation of the nasal sinuses and, since the work was experimental, we applied it without discrimination in all sorts of cases.

We were soon convinced that in all suppurative sinuses, improvement was obtained such as we had never seen in the conventional methods of treatment, and in some instances the results were remarkable.

In the first place, there was usually observed with each application a marked relief of the local discomfort and, with the continuance of the treatment, the benefit was evident in the improvement of the character and the amount of the discharge.

Especially gratifying was the observation that some cases cleared up completely, in which we had thought radical surgery was inevitable.

We will not attempt at this time to give a statistical report of our experiences, but will cite briefly two or three cases which are in themselves significant.

A woman, age about 35 years, had during the winter several attacks of severe persistent head colds, marked by heavy yellow discharge. Six weeks before I saw her, there had been an unusually severe attack, probably of influenzal origin, attended by pain in the right cheek, nasal stoppage and discharge, all of which symptoms were still present.

Puncture and irrigation of the antrum gave a very abundant discharge. On account of the prompt refilling of the antrum, the irrigation was repeated every other day for ten times. At the end, the

condition was as bad as at the beginning, and I advised the patient that a radical operation was necessary.

At this stage I began a trial of iodin fumes. The patient was at once conscious of marked relief. The suppuration began promptly to diminish and, after four treatments, completely disappeared and so remained.

A man, age 40 years, had had both maxillary antra radically operated; the right side by a prominent specialist of the city four years ago; the left, by another specialist one year ago. Both antra were freely suppurating and the patient suffered much local discomfort and was greatly discouraged.

The case presented opportunity for the experiment of testing the efficacy of the iodin fumigation by using this method on one side only and comparing it with the result obtained on the other side by simple irrigation.

The experiment was conclusive. The suppuration on the side on which the iodin vapor was used promptly cleared up, while on the other it continued as before.

Another case which in itself seemed to give fair ground for definite conclusion was that of a prominent physician, who for the past year had suffered from recurrent attacks of iritis, with disastrous consequences to his eyes. For the past six months these attacks had become more frequent, in fact they had been occurring almost regularly every ten days, always marked by red, suffused eyeballs, clouded vision and headache.

Vaccines had been given in vain, and different foci of infection had been suspected and treated without benefit.

About ten years ago the patient had had trouble with his antra and both had been radically operated. Large artificial windows were still present in the inferior meatus, offering an easy route for exploration and treatment of the sinuses.

As pus was discovered by washing out, we instituted a course of iodin fumigation, and the effect was immediate and pronounced.

Each time that the patient presented himself at the onset of the attack, application of the vapor caused immediate disappearance of the congestion and tension of the eyes, and the next day the symptoms were gone and the attack aborted.

The patient for several months now has been entirely free of the troublesome eye symptoms, and his sight has measurably improved. He reports for occasional treatments as a matter of precaution.

The first case proves that the iodin vapor treatment is effective where a fair trial of the usual method has failed; the second case,

its efficacy in comparison with ordinary methods on the other side in the same patient; and the third, the immediate benefit evident from each single application and the final success in clearing up serious complications of long standing.

The procedure I employ is as follows: If there is much pus present, the sinus is first irrigated with a simple alkaline solution for its cleansing effect. Then, with the cannula still in place, connection is made by a rubber tubing with the delivery end of the electric heater and a current of hot air driven into the cavity of the sinus, until we judge the walls to be thoroughly dry. A charge of powder, which for the average case is from 30 to 60 grains, or a half to one teaspoonful, is then placed in a perforated metal capsule which fits snugly into the barrel of the electric heater. As soon as the heat reaches a sufficient degree (about 237° F.) we will be aware of it by a small cloud of smoke emerging from the outlet tip of the heater. The latter is now again connected with the cannula in the nose and the vapor is carried into the interior of the sinus under a very gentle current of compressed air.

Notwithstanding what seems to be very striking successes, I do not wish to be understood as claiming that all and every kind of suppurative sinus can be definitely cured by the method described.

Cases in which there is deep or extensive necrosis of the bony walls, and in which the sinus interior is filled up with thick growth of polypoid granulation are proper for a radical exenteration and probably cannot be otherwise cured.

There are, however, a great number of cases just short of this, cases of persistent suppuration, that by the usually accepted criteria are regarded as ripe for operation, in which surgical measures may be avoided.

When we consider the long established reputation of iodin therapy and when we consider that special therapeutic virtues are inherent in nascent iodin, it ought not to surprise us to see excellent results from the iodin fumigation of the suppurative sinus.

The merit of the treatment here outlined consists we believe, firstly in the employment of a method of producing nascent iodin which can at the same time be turned to advantage in securing the most favorable condition for its beneficial effect, namely, a dry field and secondly, in the devising of a practical procedure for delivering the fumes to the interior of the diseased cavity.

Iodin is an ancient and honorable agent with a splendid service record in the healing art of more than 100 years' standing. Taken all in all, we doubt if there is another drug which has to its credit so many and such brilliant successes in combating diseases of the

human race. Why discard a remedy like this, whose excellent healing qualities we know by testimony of so many disinterested witnesses, to take up with new remedies that we do not know at all, and which we are asked to accept by their interested sponsors?

Let us rather cling to the old, seeking new application and finding new possibilities.

1606 Twentieth Street N.W.

A CASE OF GIANT CELL TUMOR OF THE SEPTUM NASI.*

DR. HENRY S. WIEDER, Philadelphia.

The following case report is presented because of its uniqueness, since giant cell tumor attacking cartilage in any situation is a rarity as far as the writer's knowledge and experience is concerned. For this tumor to attack the septum of the nose is especially rare. In the course of almost thirty years' experience in pathology and in the perusal of literature in the field of rhinology, the writer does not recall ever having encountered a single case. These tumors attack bone in particular and give rise to cystic growths, are often found along the gum margins in the form of epuli, or, finally, may be found in the tendon sheaths. Formerly they were considered a slow growing form of sarcoma but in recent years it is recognized that but few of them are malignant in character. Therefore, it is now customary to refer to them as giant cell tumors and not sarcoma.

The body of the tumor usually is composed of rather large spindle cells, having multinuclear giant cells resembling the foreign body giant cell thickly interspersed throughout the tissue. They do not tend to give metastasis and if thoroughly removed show no tendency to recur. This, however, is not an absolute rule, since occasionally, though very rarely, one proves malignant. Therefore, X-ray treatment is advisable at all times in the handling of this tumor.

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*Read before the Philadelphia Laryngological Society, Dec. 1, 1931.

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Case Report: S. G., age 27 years, white female, while rising from a stooping position accidentally hit her nose violently against a door which was ajar. Though the nose was extremely painful, she sought no medical aid but allowed it to heal of its own accord. Several weeks later obstruction was felt in the left nostril, this being particularly noticeable because she suffered from atrophic rhinitis and would occasionally introduce her little finger into the nostrils to loosen crusts. On one of these occasions she encountered a small nodule on the left side of the septum and reported to the writer for examination.

Inspection of the nose revealed typical atrophic rhinitis, with a small round sessile nodule about three-eighths of an inch in diameter, projecting from the cartilaginous portion of the septum. It was thought that this had probably been produced by irritation from the accident and that it partook of the nature of callus. It was therefore removed with a cold wire snare, with no great difficulty. The specimen was examined microscopically more from the standpoint of curiosity than from suspicion of malignancy. To the writer's surprise it proved to be a typical giant cell tumor, as was confirmed by two competent pathologists. The patient was referred to Dr. William L. Clark for X-ray therapy and was given a series of cross-fire treatments. It is now thirteen years since the removal of the tumor and there has been no sign of recurrence, but the area of the septum from which the tumor was removed shows a dead white color on both sides, unlike the remainder of the septum. Whether this is due to a change in the mucosa as a result of the X-ray treatment causing increased fibrosis, or is due to destruction of the vessels in that area, the writer is unable to say.

2106 Spruce Street.

THE USE OF CALORIC NYSTAGMUS ELICITED BY WEAK STIMULI AND ITS PARTIAL PHENOMENA IN OTONEUROLOGIC DIAGNOSTICS.*

DR. TIBOR GERMAN, Budapest.

The physiology of the labyrinth has come in for a far-reaching improvement in the last decade. The starting point of this period of advance is undoubtedly the work of Kobrak, begun in 1918. He was the first to show that the terminal apparatus of the vestibular nerve was susceptible to being put into an irritative condition by means other than the routine procedures of calorization and rotation. We are furthermore indebted to Kobrak—only in order to mention some of his classical statements—for having made us acquainted with the notions of nystagmus disposition ("Nystagmusbereitschaft") and of labyrinth sensitization; he was the first to give a detailed description of the nystagmus-inhibiting or damping action of stronger stimuli. His vascular theory on nystagmus led the scientists who believe in the physical basis of nystagmus to make further and more intense research in the subject. On his work rest the later researches of the Vienna, Frankfort, Cologne and German Prague Universities and many authors (Brunner, Grahe, M. H. Fischer, Wodak, Frenzel, Veits and so forth), whose findings resulted in offering us a more exact explanation of experimental nystagmus. His work aided too, in bringing Bruening's pessimum head position out of its former only theoretical significance into the realm of practical importance in that the calorization procedure has thus been worked out into a very sensitive quantitative method; and allows the various preliminary phases of nystagmus to be evidenced individually. In the last decade falls also the discovery of the arm tonus reaction of Wodak (Bárány's pointing test is of older date), and the bilateral (simultaneous) douching first applied by Ruttin, which all of them have led to the discovery of the extremely interesting vestibular pulsion reflexes; in this epoch, too, falls the fundamental experimental work of Magnus and De Kleyn on the postural reflexes serving in its turn as foundation for the improvement of the diagnosis and pathogenesis of the otolith diseases.

*From the Otological Clinic of the R. University in Budapest (Director: Prof. Dr. Géza Krepuska).

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The core of the static functional tests lies in the calorization tests which up to Kobrak's discovery were everywhere carried out with the aid of massive douchings in such a manner that cold (27°) or hot (47° C.) water was injected into the auditory meatus under mild pressure until nystagmus occurred, whereupon estimations concerning the amount of water necessary for provoking nystagmus, then of the degree, intensity and duration of the nystagmus were done. This procedure, however was associated with a great many inconveniences for the patients as there occurred in almost all of those cases extreme vertigo, often nausea and vomiting. Another drawback of this procedure lies in the slow appearance of the reaction. Its dependability is interfered with, among other things, by the unavoidable nystagmus-inhibiting action of the massive douchings, regardless of whether it is interpreted as a reflex phenomenon elicited by the auditory meatus or, as evidenced by Grahe's researches, as a central phenomenon.

In the presence of injuries such as rupture of the tympanic membrane, fracture of the auditory meatus, petrous bone and floor of the skull, massive stimuli cannot be applied either in the form of rotation or calorization. In these cases weak stimuli give us the chance of obtaining information about the condition of the vestibular apparatus of the diseased ear by the calorization of the sound side (by way of sensitization).

Calorization by way of weak stimuli is never associated with subjective troubles, especially vertigo. Its use for clinical purposes is very simple. We have adopted this method so much more gladly as we still distinctly remember that at the beginning of this century, on the occasion of Bárány's report of having produced nystagmus by calorization, a very celebrated specialist had protested against the "inhuman" procedure with indignation. Although the events have fully proved this adversary not to have been right, and in spite of calorization having gained a footing everywhere as an important diagnostic adjunct, it will nevertheless prove gratifying to deal with a procedure apt to essentially reduce the inconveniences brought upon the patient without thereby lessening the value of the method. Calorization by weak stimuli has systematically been carried out in our clinic for two years; the technique has of course undergone several modifications, until we now have a standardized procedure which we have used for a year. It is more sensitive than mass douching and furnishes dependable and valuable data. Because its even slight variations are not to be made light of, it is applied of course first and foremost in cases in which gross functional variations are not ex-

pected. Should they, however, be present, profuse douching will still prove convenient, when weak stimuli will have failed to show the desired information. In addition, the investigations have enriched us with new facts thus far unknown in the physiology of the labyrinth and otoneurological diagnostics.

Apart from some modifications, the technique of calorization by weak stimuli follows upon the whole that described by Veits and is very simple. It is as follows:

The patient is first tested for spontaneous phenomena such as nystagmus, pastpointing and falling. If there be spontaneous nystagmus, then the direction of the gaze ought to be fixed by Bruening's otogoniometer. After the head of the patient has been bent forward at an angle of from 20 to 30°, 10 c.c. of water at from 10 to 20° C. are injected into the auditory meatus by a Pravaz syringe within five seconds. By the aid of a drain the jet is directed towards the posterior upper wall of the auditory meatus near the tympanic membrane (antrum bridge). In this position nystagmus should not occur. If it does occur, and especially towards the opposite side, the head ought to be bent forward still more; if it occurs towards the tested side, the head will have to be lifted somewhat. Sixty seconds after the end of the douching the head is bent backward 90°, which manipulation should be performed in two seconds. It takes the nystagmus some seconds to occur, this being the latent period which is estimated most exactly; then the nystagmus is always tested in the primary position behind Bartel's glasses and its duration determined. The nystagmus usually occurs in a short time, starting with a slow deviation of the eyeballs to the side under observation, and one or two irregular twitchings. After the nystagmus has ceased, the measurements are continued in lateral position till it disappears completely. An interval of from 15 to 20 minutes must be allowed between the examinations of both sides.

The individual factors allow for some comment which is in agreement with Veits, given as follows:

1. First let us say a word or two about the position of the head. The position in which the douching is made (forward bending of the head, 20 to 30°) corresponds to the real horizontal position of the external semicircular canals, since their plane, as is well known, slopes backward. This position is called "pessimum position" by Bruening, but should be called *indifferent position* (M. H. Fischer), with regard to calorization (and pulsion reflexes), an endolymphatic current cannot be produced, hence nystagmus cannot arise. It is the position from which exact examination of the labyrinth

ought to start. When the head is brought out of this position, it comes into an irritative position entailing nystagmus, the intensity of which is in the direct ratio to the distance from the indifferent position. When the head is bent backward, there occurs contralateral nystagmus (ampullo-fugal current), which reaches its maximum when the head has been brought out of the indifferent position to 90°; *i. e.*, when the horizontal semicircular canals come into a vertical position. This is the so-called *optimum position of the head.* Beyond this position the nystagmus diminishes in intensity, until the *posterior indifferent position* is reached, which places the semicircular canals again in horizontal orientation differing from the anterior position by exactly 180°. Then if the head is bent backward beyond this posterior indifferent position the nystagmus, similarly to what happens with the head inclined beyond the anterior indifferent position, passes to the side submitted to the douching (ampullopetal current).

2. The injected stream ought never to enter the auditory meatus at random, but, by means of the head mirror and speculum, should be directed exactly towards the posterior upper wall, the so-called antrum bridge of the auditory meatus; it is only in this way that the *change of temperature* can assert itself in the external auditory meatus *quickly and integrally.* It is not sufficient that the amount and temperature of the water are steady; care should be taken also to keep the direction of the injected stream steady for although the change of temperature involves the internal ear, it will nevertheless prove different in regard to the extent and duration of the nystagmus. Caution is necessary when dealing with tortuous auditory meati because of this interference to the stream.

3. In our opinion the *optimum duration of douching* should not exceed five seconds (one pressure on the piston per second) (Veits performs the douching within seven seconds). This length of time should always be observed, because this factor, too, changes the temperature of the internal ear. It is only in such a manner that dependable comparative values are obtainable as to both sides of the affected patient, in comparison to normal individuals. As to the temperature of the water, we prefer 20° C. because cooler water may elicit unpleasant subjective symptoms and exert a nystagmus inhibiting action. The amount of water to be used has been determined at 10 c.c. since douching with smaller quantities of water or with warm water of over 20° C. produces too short a nystagmus for the differentiation to be properly estimated.

4. This suggests the question as to why it is deemed advisable to wait a while after the douching is done and why this interval ought to be just 60 seconds; this procedure is at variance with that of Veits, who includes the duration of the douching itself under the waiting time. This question can be answered by the fact that—according to Voelger, Dohlmann and Schmaltz—the withdrawal of heat experienced by the external semicircular canal arrives at its maximum in about one to one and one-half minutes, wherefore the position of the head must be changed during this period in order to attain the maximal reaction. Under 60 and over 90 seconds the potency of the stimulus is substantially decreased, so by selecting a waiting time of 60 seconds we have chosen the *minimum space of time in an effort to attain the maximum stimulus.* Comparative values are only dependable when the same technique is always strictly observed, otherwise differences could arise in which the irritability could not be determined.

Another question arises as to why the injection is not at once made in the optimum position. For the purpose of a better understanding of this subject let us say a word or two about the latent period following irrigations in the irritative position. It consists, properly speaking, of two components: the one is at work till the cooling extends from the auditory meatus into the labyrinth and is dependent on physical factors; the other, however, comprises the span from the onset of the endolymphatic movement up to the occurrence of the nystagmus and is controlled by factors of purely physiologic nature. The first mentioned component is the thermal phase of the latent period, Frenzel's so-called "thermal time threshold"; the latter phase, the "latent period proper." When the test is carried out in the irritative position, there is no segregating of each from one another; however, when the examination is begun in the indifferent position, then the contents of the semicircular canal have cooled conveniently, after one minute's waiting, while the flow of the current is impeded until the semicircular canal has left its indifferent position. During the time it takes to bring the head into the optimal position, the thermal phase of the latent period has already passed and the next phase of the latent period corresponds already to the *pure physiologic "latent period proper."* As the thermal phase of the latent period depends chiefly on extralabyrinthine factors (pneumatization variants), it has only a poor claim to our interest in the rational of the reaction; the thing we are interested in is the phase of the physiological "latent period proper" and that can only be estimated in the aforesaid manner.

5. The change of position which the head undergoes when it passes from the indifferent to the optimal position is always performed within the same space of time (two seconds), because the speed of the change of position acts materially on both the latent period and the duration of the nystagmus. With a quick change of position the latent period is shortened and the duration of the nystagmus prolonged, and *vice versa;* when the head is brought slowly from the indifferent position into the irritative position, the latent period is prolonged and the duration of the nystagmus shortened. This can be pushed so far that the head can be quite cautiously placed, or smuggled into the optimal irritative position without nystagmus occurring at all. This fact does not allow of a purely physical explanation, since the speed of the endolymph current is of necessity independent of the duration of the change of position; it seems, however, that, setting aside habituation, provocation of the caloric nystagmus is not so much a function of the current speed but rather one of the increase in speed attained in the unit of time.

6. The nystagmus is tested in primary position with Bartel's glasses applied, because in this way all of those troubling agents are eliminated, which on the one hand are due to optic factors (fixation), and on the other hand to the enhanced tonus of the eye muscles, the strong convex glasses precluding fixation, which fact in turn prevents the muscles of the globe from being strained in the primary position. Furthermore, any movement is seen magnified through convex spectacles, so that the eye of the observer is less quickly fatigued. The possibility of reducing eye tonus to a minimum makes up for the slight drawback that nystagmus occurs in the primary position later than it does in side position. When the twitchings in the primary position have already come to an end, they are still extant in side position (we purposely avoid the term of side-look). Particular importance has quite recently been attached to the test of both this circumstance and the measurement of the corresponding length of time for reasons that we shall later attempt to explain.

7. The duration of the nystagmus is calculated from the moment the eyeballs begin deviating from the primary position, *i. e.*, from the onset of the slow component. The latter is occasionally followed by some irregular twitchings, which frequently, in gradual transitions, pass into the quick component. In contrast to the douchings carried out in the irritative position the double twitchings as observed by Kobrak and Grahe hardly ever occur. It is therefore easier and safer to estimate the slow component, which from the point of view of appreciation of the vestibular function is of greater importance

because of the quick component depending on the function of higher centers. Recently some authors (Frenzel and Veits) measure the number of twitchings instead of the duration of the nystagmus. After a fair trial we found that both methods gave rather uniform results. However, we prefer our own method because we believe that the chance of error is less. When the length of time is measured from the onset of the slow component, the initial errors can be reduced to a minimum; although the end of the nystagmus in the primary position is always somewhat doubtful, as there occur at times one or two more jerks after a longer interval, still when patient is looking sideways there is an easily perceivable nystagmus which is measured beyond the duration of the typical twitchings as long as there are still movements of the eyes, *i. e.*, till the slow component ceases. This occurs as a rule without transition and is easily perceptible. We feel also that this point of view is a more rational one for the estimation of the vestibular function, since, as a matter of fact, it is the quick component which is counted in the computation of the twitchings; besides, allowance is made for the quantitative character of the procedure by the more exact timing.

When an intact vestibular function is tested with the above mentioned method, the results are fairly consistent, revealing a latent period of from three to five seconds; a duration of the nystagmus in primary position of from 80 to 110 seconds, with addition of further 8-14 seconds in side position. The tests undertaken on one and the same individual at various times result in findings identical almost to the second. An instance may illustrate these statements.

Clinical Diagnosis (Mrs. J. B.): Otosclerosis with hyperesthesia of the cochlear nerve. The irrigation of the right ear, carried out in the typical manner, yielded the following results:

		After 20 Min.	*After 40 Min.*
Latent period	4 sec.	4 sec.	4 sec.
Duration of the nystagmus in primary position	81 sec.	84 sec.	83 sec.
In side position	10 sec.	9 sec.	10 sec.

The physiological limits, however, lie probably below and above these figures, so that in the primary position a duration of the nystagmus of 70 and 120, respectively, has to be considered still normal; a lowered stimulus-threshold, hence exaggerated irritability can therefore only be spoken of when lower and higher values are found. Valuable information to the point can also be given by the latent period, because, as can be inferred from what we have stated above, the latent period is inversely proportional to the irritability, *i. e.*,

with increased irritability the latent period observed is shorter, with lowered irritability longer than the normal latent period. The first degree nystagmus tested in side position shows in regard to the duration only very rarely more noticeable departures.

At the beginning of this paper we have mentioned that by way of weak stimuli we have succeeded in individualzing also the several previous phases of nystagmus. The matter concerned here is that sphere of excitement which can hardly ever be brought on by mass douchings, namely, the spheres of the slow component and of the initial twitchings. They can, however, be detected fairly frequently by douching with 10 c.c. and conducting the test in optimal position; it is for this very reason that the appearance of the slow component is for us indicative of the outset of the vestibular stimulus. The weaker the applied stimuli are, the more easily the single phases can be made amenable to separate observation; sometimes 3 c.cm. of water at 35° C. are enough to allow of attaining the desired object. Within the scope of our technical procedure we have attempted to replace smaller amounts of water by slight stimuli in bringing the head from the indifferent position, not into the optimal one, but in lifting it only a few degrees. In some of the cases, however, there appeared no nystagmus after a latent period of variable length (from 8 to 15 seconds), but there occurred notwithstanding a *slow, pendular excursion of the eyeballs* of from 20 to 30° to the right and left from the primary position, which phenomenon came to a standstill after this procedure had been repeated three or four times. The greater the distance from the indifferent position of the head, the less distinct was this phenomenon; however, it was also present during the indifferent position of the head, though we had the impression that it manifested itself as such more rarely and with lesser intensity than it did when the head was tilted beyond its indifferent position.

J. K., academician: With intact condition of the tympanic cavity had slighter fits of vertigo. The cochlear nerve was sound. The vestibular test by means of weak stimuli yielded the following data: Findings on either side approximately equal; latent period, five seconds; primary position, 90 seconds; side position, 12 seconds. At the next test the head was lifted 10 degrees; after 10 seconds there occurred four times a distinct pendular deviation of the globes, without subsequent nystagmus. The test was repeated at the end of half an hour, at which time the pendular movements again occurred; on bending the head forward there occurred two or three doubtful excursions.

The physiological interpretation of this phenomenon is difficult, because it is quite inconceivable that the sphere of the slow component cannot be safely estimated by the excursions; in this case one could only expect to see an excursion to the one side, perhaps a deviation of the eyeballs; but not a pendular movement also to the other side. The assumption that we no doubt have to deal here with a vestibular phenomenon is also supported by the fact that it could at no time be seen in cases with functional deficiency of the vestibular apparatus. It is probable that owing to a minimal movement of the endolymph, perhaps better elicited by our test method than it was by the caloric "minimum" — stimulus in erect position of the head — the sphere of the low component was successful in being isolated to so large an extent, that it was not at once followed by the sphere of the initial twitchings, on account of the stimulus threshold necessary for the production of the quick component not yet having been attained. Nor can the excursion to the other side correspond to the quick component on account of its rate; besides were the quick component concerned, one would be justified in expecting a greater activity of same, which, as we have seen, was not present, since the excursions ceased completely after repeating two or three times. It looks, therefore, as if in some cases it has been possible to obtain that phase of the slow component in which *specifity of direction* is not yet present, but owing to sensitization of the center—as Kobrak could evidence by water at 35° C. in regard to nystagmus—the slow component can be produced as characterized by its unspecific direction towards both sides. Its appearance prior to the occurrence of the typical contralateral nystagmus and its correspondence with the homolateral and then bilateral nystagmus. Analogous to the slow component is perhaps that pendular excursion of the eyeballs found under deep anesthesia by Borries, Rejtoe and others, and quite recently observed by Kelemen and Klimko under nitrous oxid anesthesia. This phenomenon was by these authors, too, interpreted as a vestibular reaction and, similarly to the eye movements detected by us, always resulted in associate motions in such a manner that the globes never reached their extreme positions and the excursions to both sides occurred at a uniform rate. Still greater is the resemblance to a case of the last mentioned authors in which similar ocular movements were elicited by *calorization during anesthesia*, further to a case of Rosenfeld, in which the calorization carried out in morphiascopolamin (twilight sleep), likewise produced pendular movements of the eyeballs. It seems likely that the central sensitization in these cases has also been caused by the anesthetic itself.

Now we have arrived at that point where we intended giving an explanation of why the first degree nystagmus is submitted to testing, although the latent period and the measurements undertaken in primary position about the vestibular hyper- and hypoesthesia give sufficient information. In case of purely peripheral lesions one can indeed be quite contented with the test in primary position alone; if, however, a central affection is suspected, then an examination in extreme position should also be made. At this examination attention should be paid not only to the duration, but to the difference between both eyes as to duration and intensity of the nystagmus. The differences presented thereby, however, are not of vestibular origin, but the manifestations of a centrally prevailing disparity in the tonus of the eye muscles (Kobrak); hence the phenomenon can be conveniently utilized with a view to side localization. Since the central vestibular hypersensitivity can be uniformly elicited on either side, and the fainter the set-up stimuli are, the difference prevailing between both sides ought to be determined at the douching of either of the ears. It will, therefore, prove most beneficial to test the side nystagmus after the second degree nystagmus has cleared up, because by this time the stimulus is already attenuated on the one hand, and the course of the test in the other is simplified because repetition of the calorization can be dispensed with.

T. J., a girl, age 17 years (referred from the nerve department of the policlinic): For years she had complained of cephalea, bad sight and poor hearing, which continued to progress in process of time. Cochlear nerve: on the right, 0; on the left, whispered voice at 10 c.m. (4 inches). Weber not lateralized, Rinné on the left, positive; bone conduction strongly shortened on both sides. The caloric test by weak stimuli resulted indifferent; then, in optimal position, the finding on the right was, indefinite twitchings in primary position, and nystagmus during 12 seconds in side position. *The right eyeball showed the more vivid twitchings.* Left side: after a latent period of two seconds, there occurred a nystagmus in primary position for 15 seconds, *while the right globe repeatedly presented more intense twitchings of several seconds longer duration.* The clinical diagnosis was: brain tumor (pontocerebellar) of the right side. The autopsy revealed the presence of multiple neurofibromata the size of beans, soldered up with the nerves and also scattered over various areas of the dura, involving also both inner auditory meati. *On the right side there was a cerebellopontine tumor the size of a walnut.*

In order to decide whether the seat of a vestibular lesion is central or peripheral it is a matter of course that besides the cochlearis test,

the latent period and the duration of the nystagmus, or rather the interrelation of these two factors also must be taken into consideration.

Let us add some words on the nature of the nystagmus observed in the course of our experiments. The nystagmus elicited by profuse douching is always horizontal-rotary, whereas that elicited by weak stimuli is without exception *purely horizontal*. The horizontal-rotary nystagmus elicited by profuse douching grows gradually horizontal when the subject under examination looks to the opposite side, and gradually rotary when the patient looks to the irrigated side (Borries). Weak stimuli produce no alteration in the character of the nystagmus. No matter in which side position the test is performed, a horizontal nystagmus always results; probably because the change of temperature in the frontal semicircular canal, or rather in its ampullar portion, occurs only to a very low degree and the endolymphatic current of the frontal semicircular canal is interfered with by the optimum position of the external semicircular canal. For all that we have been able to observe behind Bartel's glasses, the nystagmus is in some measure of rotary character in part of the cases in the initial phase of the reaction; here one has to deal with hypersensitive cases with short latent periods and long duration of nystagmus. In side position, however, the rotary character of the nystagmus ceases likewise. If the *maximum of irritability* is fully utilized, due to the advantages afforded by our technique, *i. e.*, if the change of the position of the head is not changed within two seconds as usual, but in a shorter time—let us say in half a second; or, to be short, in a fulminating manner, then the *previously rotary character of the nystagmus will be more pronounced or disappear in side position*. The repetition of this experiment has yielded analogous results.

J. S., day laborer, age 53 years: Five days before he was seized with vertigo and became unconscious. He complained of severe headache. Blood pressure, 220 m.m. Hg. Serpentine veins on the fundus oculi. No pareses. No spontaneous vestibular symptoms. Cochlear nerve: whisper on both sides at some three meters; Weber not lateralized; Rinné positive; bone conduction shortened on the right by 10 seconds, on the left by 14 seconds. Vestibular nerve: weak stimuli in at first indifferent, then optimal position of the head showed, on the right a latent period of two seconds, horizontal (rotatory) nystagmus in primary position for 220 seconds, in side position for 12 seconds; in the left the latent period was two seconds, the duration of the nystagmus in primary position 240 seconds, and in

side position 14 seconds. Quick postural change of the head resulted in immediate occurrence of horizontal-rotatory nystagmus in both the primary and side positions. The calorization of both sides led to the same result. The clinical diagnosis of brain tumor was made.

The explanation of part of this phenomenon appears to be fairly simple, in that with the hypersensitive vestibular apparatus an endolymphatic current and consequently the corresponding current rate (ampullopetal current) is produced also in the frontal semicircular canal, rotatory elements being thereby added to the prevailingly horizontal component of the nystagmus. But how can the fact be explained that, on the one hand, the rotatory character of the nystagmus does not come to a standstill in side position, although this is the case when profuse douches are applied; while, on the other hand, there occurs a transformation to a horizontal nystagmus? For the interpretation of this phenomenon we must avail ourselves of Bárány's latest theory on the interaction or rather mutual inhibition of one kind of nystagmus by the other. It seems, however, more plausible that in the case in hand we are not dealing with such an interaction, but on the contrary with the fact that profuse douches, in addition to their nystagmus-inhibiting effects, also possess the capacity of exerting an influence on the mode of manifestation of the nystagmus, especially in side position, which is also nystagmus-inhibiting in itself. When weak stimuli are set up, these impediments are absent, *i. e.*, they cannot assert themselves. If we take into consideration that in all of our cases concerned, as well as in the above cited one, there were *central affections*, it would seem that this explanation is so much more acceptable that there will hardly be any doubt about the central origin of the inhibition. The complete absence of the inhibition is therefore suggestive of the presence of a central lesion and it may well be (the investigation of further cases will have to find that out), that this phenomenon will prove a valuable adjunct to the very scanty armamentarium of the otoneurologic diagnostics of intracranial diseases.

SUMMARY.

In the caloric test of the vestibular apparatus the tests carried out by the aid of weak stimuli are superseding the profuse douches more and more; the first mentioned procedure is simpler, and besides more perfect and more sensitive than is the latter. Probably the most important step has been the modification according to which the douching is performed in indifferent posture of the head and the test for the reaction is carried out in the optimum position of the head. The advantages of the procedure are as follows: it causes neither

vertigo nor subjective complaints and, should there be a traumatism, may be applied by way of sensitization from the other side. Owing to the elimination of inhibitory factors the results afford a clearer picture of the irritability of the vestibular nerve than do the profuse douches, the latter disclosing rather the peripheral differences at the expense of the differences in the central tonus. The physiological portion of the latent period can be isolated, which is of prominent importance for the appreciation of the vestibular irritability. Further, the procedure furnishes materially more exact results because it admits of a test without straining the extraocular muscles. It is irremissible to repeat the tests several times in every case, but always with exactly the same technique. The procedure by no means dispenses with the use of the other tonus reactions (pastpointing, arm tonus reaction, pulsion reflexes); however, profuse douchings will prove to be necessary only in case of severe labyrinthine lesions.

The individual phases of the slow component can be easily isolated by certain manipulations.

Thus we can consider otoneurologic diagnostics improved first from the point of view of side location, in that not only in the primary position of the eyeballs do differences in tonus occur but also on the tests made in extreme side position; and second, from the point of view of central or peripheral vestibular hyperesthesia, in that in any position the nystagmus has rotatory character, when the head is abruptly tilted backward.

ACOUSTIC REFLEXES ON THE MUSCLES OF THE MIDDLE EAR (PRELIMINARY COMMUNICATION).*

Dr. R. Lorente de No, St. Louis.

The existence of some muscular reflexes to acoustic stimuli has long been known. It seems, however, that these reflexes are not constant and, therefore, cannot be used as an indicator in making an accurate study of the hearing ability of animals. The desire to determine the physiological properties of the different centres and pathways involved in central hearing has led us to the study of the reflex activity of the muscles of the middle ear. It was thought that if some constantly appearing muscle-reflex similar, for instance, to the reflexes of the eye muscles, to excitations of the labyrinth could be found, these reflexes would give the desired information as to the presence of hearing after operations. It appeared logical to begin with the muscles of the middle ear (m. tensor tympani, m. stapedius) of which contractions in response to acoustic stimuli have been described many times before. In the literature there are two fundamental papers (Kato, 1913; Mangold, 1914) containing the old literature on the subject and reports on experiments on animals and even on human beings.

We approached the experiment with great skepticism, because the general idea is that contractions of these muscles occur only when the tones are very intense and, therefore, the function should be to protect the ear against intense tones. Kato even claims that if the muscles of the middle ear are cut, the ear is much more liable to damage by intense tones than normally. On the other hand, modern researches with the help of the oto-microscope have failed to demonstrate movements of the drum membranes and, therefore, the activity of the muscles of the middle ear has not been considered as being a possible cause of error in the study of the action currents of the acoustic nerve. In spite of the doubts with which we approached the subject, the results could not have been more encouraging. The technique was as follows:

The external auditory canal and the drum membrane were extirpated and the malleus and incus removed. It was then possible to

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examine, under the microscope, the tendon of the muscles tensor tympani and the stapedius. The animal used was a rabbit under local anesthesia. It was found that whenever a tone reaches the ear, the muscles contract and it is not necessary that the tone be a strong one. Rather weak tones are sufficient to produce visual reflex contractions of both muscles. Although we cannot state it with absolute certainty, we think that the two muscles react each to different kinds of tones, the muscle stapedius beginning to react to low tones and the muscle tensor tympani giving stronger reactions to the higher ones. But the most interesting thing is that these muscles follow the inflections of speech, thus indicating that they are able to respond to changes in intensity or pitch of sound. If words are spoken, the muscles reproduce the changes in inflection of the voice in very much the same way that the strobilion would do. Although the reflex reaction of the muscles of the middle ear is going to be the subject of intensive studies in this Institute, two reasons justify the publication of this preliminary note:

1. Since the reflex reaction of the muscles seems to be a constant one, even to weak tones, there is no doubt that these reflexes constitute an indicator of the hearing ability of the animal and, therefore, enable us to study the physiology of the cochlea and hearing centers in the medulla oblongata.

2. Since both muscles in their contractions must develop action currents, it would be helpful in the study of the action currents of the acoustic nerve to extirpate the muscles of the middle ear.

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THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of Feb. 17, 1932.

(Continued from September issue)

DISCUSSION.

DR. MARION B. SULZBERGER: As the skin and the mucous membranes are apparently the most frequent sites of hypersensitiveness in human beings, it would be extraordinary and astonishing to find that the mucous linings of the sinuses, the nose or the ear were exempt from allergic manifestations. The symposium as so ably presented by the essayists of the evening shows that, as was to be expected, this is not the case. I believe that the consideration of the allergic responses of these areas is but at its beginning. This conception represents a distinct broadening of viewpoint which will inevitably lead to advances in otolaryngology. I warmly approve of the definition of allergy as given by Dr. MacNeal and have always used the word in this sense. This definition not only has the advantages of clarity, precision and logic but also adheres to the original meaning of the word when first employed by v. Pirquet and Schick. We must, however, bear clearly in mind that calling a manifestation allergic merely points to a path for further study and by no means implies that a goal of etiological or pathogenetic research has been reached. If we bear this in mind, we shall avoid the dangers which might arise from the too liberal generalization of the allergic conception.

Here, as in all other manifestations of hypersensitiveness, the situation is complicated by various possibilities. I should like to recapitulate some which seem to me the most important:

1. The nose, throat, ear, sinuses, mastoids and so on may be the seat of primary infections. From these foci products may circulate which act as allergens and cause the appearance of classical allergic manifestations, such as asthma, hay fever and urticaria. As Dr. Pease has pointed out, these foci may be insignificant and still cause severe general manifestations. As illustrations, we need only think of such a minute form as an apical infection of a tooth and its possible effects. I myself often see this point made apparent in the cases in which a small, scarcely visible discoloration of a toenail harbors fungi and sometimes causes severe generalized and refractory skin eruptions and general illness which may persist and incapacitate for years.

2. Infections and the resulting injury to the mucous membranes may result in a heightened permeability which facilitates the absorption of extraneous allergens; for instance, air-borne pollens and epidermals. This increased absorption then produces sensitization and allergic disturbances.

3. Allergens, either air-borne or hematogenous, may cause primary allergic reactions in the nose, sinuses, ears and other organs. Repeated reactions of this type lower the resistance of the tissues, which then become secondarily infected by invading organisms. (This group is perhaps one of the largest.)

4. Infection of the mucous membranes may weaken them in such a manner that allergic reactions due to circulating or air-borne allergens become localized in the areas which are or have been infected.

5. The organs of the ear, throat, nose and paranasal sinuses may be shock-tissues in which primary allergic reactions take place simultaneously with or independently of the reactions in other shock-tissues, such as the bronchioles in asthma.

Some of the conditions I have outlined have already been proven to exist; for example, the fortunate coincidence of an asthmatic attack occurring in a patient whose antrum had been filled with lipiodol enabled Arthur Proetz to show that the thickness of the mucous lining of the antrum increased 1 c.m. during the asthma attack, which had been caused by feathers.

E. R. Lewis and Hansel, Hastings, Feinberg, Coates and Ersner and others have called attention to allergic mechanisms in otolaryngological conditions, including affections of the middle ear, the nose and sinuses.

All of us who have watched the typical course of anaphylactic shock in guinea pigs have noted the well known and remarkably constant symptoms of sneezing and rubbing and pawing at the nose which precedes death. W. E. Murphy most recently reported interesting results from the histological examination of the nasal mucous membrane in these animals. He observed eosinophilia, congestion and particularly petechial hemorrhages in six out of eight animals dying of shock.

It may surprise some of you that a determatologist undertakes to discuss these matters. This is explained by the fact that the skin has always offered the best opportunity for the study of allergic conditions. This holds true both as to conditions occurring in the skin and those of other organs.

Fortunately, the skin is in most cases an excellent indicator of the state of sensitiveness of other organs and it is for this reason that skin reactions prove of value in so great a variety of conditions. I am sure it will suffice for me to mention the Schick reaction, the Dick test, tuberculin and trichophytin reactions, and the usual asthma and hay fever tests to illustrate this point.

There are, of course, many different types of skin reaction and methods of testing. While there is no doubt that these require much further study, I believe that the immunologically trained dermatologist is perhaps the best equipped to corrolate and evaluate skin test and skin response in their relationship to clinical findings.

It seems to me likely that the urticarial type of reaction will be found of value in the acute primary allergic responses encountered in otolaryngology, which are analogous to the asthmatic attack. This form of reaction is usually due to inanimate allergens, such as pollens, foods, epidermals, etc. The late type of papular inflammatory response will probably be more useful in chronic catarrhal conditions and sinus, mastoid and middle ear affections in which bacterial infection plays a major role. The prototypes of this form of skin response are the tuberculin and trichophytin reactions; in general, it may be said that bacterial and fungus allergens are those which most frequently cause this form of late reaction.

As we are in complete ignorance of the relation of eczematous skin reaction to otolaryngological allergies, I have no opinion as to whether the patch test will here prove of value, but I believe it should be tried. Of course, variations of procedure will probably be necessary, such as testing the mucosa itself or employing fat, lipoid and carbohydrate fractions. I have recently demonstrated not only eczematous reactions and trichophytin but to tuberculin as well, and similar findings have been reported by Ravant, Ramel and recently Nathan.

This brings me to the first of two points which I should like to stress before I close my remarks.

The one is that in entering the comparatively new field of sinus and ear allergies, it is possible that we must widen our search for allergens as certain organs or parts of organs seem to have a tendency to become susceptible to particular types of allergens. For instance, the substances causing the epidermal type of hypersensitiveness known as dermatitis venenata are entirely different from those causing urticarial reactions of the skin, the former usually simple chemicals, such as dyes, antiseptics, etc., the latter usually the food allergens.

The second point is that the new conditions here under consideration may require new methods of desensitization therapy. The intradermal method of immunization has been found successful in many conditions. Tuft recently reported its success in typhoid immunization, and more recently still, Corbus used it with good results in gonorrhea. I have found that the intradermal injection method is an excellent desensitizing procedure in ragweed dermatitis and more especially in the chronic eczematous conditions due to fungi. Dr. Wise and I are publishing a short report on this subject.

If one can judge by analogy, I should expect the intradermal method to prove of benefit in the chronic and infectious otolaryngological conditions and I have reason to believe that it may be an improvement on subcutaneous de-

sensitizing therapy, even in acute noninfectious allergic diseases, including the classical asthma and hay fever.

All in all, a fascinating new field has here been opened and I wish to compliment and thank the essayists for their stimulating presentation of a subject which offers such splendid opportunities both for further investigation and practical results.

DR. HORACE S. BALDWIN: I have enjoyed the papers presented this evening and am grateful for the opportunity of discussing them. I shall limit my discussion to the field of bronchial asthma and its relation to otolaryngology.

I have found it of value to consider bronchial asthma in the light of a complex phyhiological reflex. If this is done one is impressed with the many and varied stimuli to attacks. These can be divided into: 1. The allergic stimuli, the determination of which is obtained through means of skin testing.

2. Respiratory infections involving particularly infections of the accessory nasal sinuses.

3. A large group of miscellaneous factors, including atmospheric conditions, irritative fumes and odors, structural changes in the nose, psychoemotional states, overexertion, fatigue and so forth.

If the asthmatic is studied from the standpoint of his stimuli to attacks we are less likely to allow ourselves to be unduly swayed in one direction or allow our energy and investigative work to be confined to any one particular field, whether it be the nose and throat or specific allergy.

I might say that at the Cornell Clinic we have worked in close co-operation with the nose and throat department. The greatest help we have from this department is in the treatment of sinus conditions, the removal of polyps, and in children the clearing up of adenoid conditions. Often children who have had their tonsils and adenoids presumably removed have subsequent attacks and we have found some of the adenoid tissue remaining. When this is completely removed the children are often improved. One important point to remember is that the function of the nose and throat specialist in the treatment of bronchial asthma is not only to remove foci of infection but to moderate the irritability of the nasal tissue which is so often found. Dr. MacNeal stated that anatomical and constitutional factors influence susceptibility to disease. In asthma and in vasomotor rhinitis this fundamental law should ever be kept in mind.

Dr. Ramivez in his remarks on the various stimuli which may bring on an attack made an important contribution and his remark on the importance of the intestinal tract should stimulate further work along this line.

The allergist may find that the skin in a particular patient reacts to a number of different foods. It is dangerous to cut these from the diet unless some definite evidence is found that these foods are concerned with attacks. If this is not done, often the patient suffers from malnutrition and poor general resistance. The welfare of the patient should not be sacrificed to the diet.

I am in agreement with Dr. Pauley's remarks. They coincide with our findings at Cornell.

Dr. Spain's remarks were very clear, concise and helpful. I am of the opinion, however, that one must not only consider these patients asthma as a result of sensitivity and respiratory infection, but that the large group of nonspecific stimuli already mentioned should be taken into consideration.

What Dr. Donaldson has said on nonspecific therapy is very interesting. In his hands I do not doubt that great good may often be accomplished. In general, however, I think that the danger in nonspecific therapy is in substituting it in place of the intellectual effort necessary to find more specific causes for some of these conditions.

DR. H. CLARK GROVE: For the past 18 months at the New York Hospital we have been conducting a special nose and throat clinic in conjunction with Dr. Cooke's allergic clinic, so that we have had an ample opportunity to observe the various allergic manifestations in the nose and throat. I think this relationship between immunology and rhinolaryngology has been presented very strongly by our speakers tonight and we should all derive additional knowledge from their papers.

There are a few observations made in our clinic and previously presented before the Section of Medicine, Jan. 19, 1932, which I think are worthy of calling to your attention. I shall confine my remarks mainly to the infective type of asthma or the second group, as defined by Dr. Spain. This group should interest all rhinolaryngologists very much because it is the type in which nose and throat infections play the most important role. The frequency of its manifestations is more readily recognized when we look at some figures collected by Dr. Cooke and presented before the Society for the Study of Asthma and Allied Conditions, meeting in Atlantic City last May. From birth to 3 years of age, 50 per cent of our cases were atopic (Dr. Spain's first class), while the infective cases were 31 per cent and the combined type 19 per cent. From 3 to 5 years, atopic 59 per cent; infective 17 per cent and combined 24 per cent. From 5 to 10 years, atopic 68 per cent; infective 19 per cent and combined 13 per cent. Now, if we take adults, from 30 to 40 years of age, the atopic cases constituted 37 per cent, infective 43 per cent and combined group 20 per cent. From 40 to 50 years, atopic 28 per cent, infective 59 per cent and combined 13 per cent. In other words, as the individuals grow older the infective asthma becomes more frequent, which also coincides with the increased occurrence of hyperplastic sinusitis.

Dr. Cooke and Dr. Vander Veer, in 1916, as mentioned by Dr. Spain tonight, showed the importance of heredity in atopic asthma. Recently Dr. Cooke has investigated a series of infective asthmas with the following results: Children from birth to 10 years (188 cases) showed a positive antecedent history in 83 per cent, while the atopic was 67 per cent and the combined type 70 per cent. In adults, 30 to 40 years, the infective type was 34 per cent positive, the atopic 30 per cent and the combined type 30 per cent. What we would like to do now is to study a group of cases of pure sinusitis without any asthmatic symptoms to determine any hereditary tendency in this disease and compare them with the above figures obtained from allergic individuals.

In studying a series of children with asthma we have found that the lymphoid tissue on the posterior pharyngeal wall is a very important focus of infection. I agree with Dr. Pauley and Dr. Baldwin that the adenoids are often the main source of infection and should be thoroughly removed, but infected lingual tonsils and this lymphoid tissue are just as important. We believe this lymphoid tissue, which is present as red plaques, bands or isolated nodules, is such because of repeated inflammatory processes and not a simple compensatory hyperplasia. We have had all of our cases X-rayed for sinus involvement but not a sufficient number have showed enough pathology to make us think that a postnasal discharge or activity of this tissue as a gland bulwark against infection has caused the hyperplasia. Most of these cases have showed an allergic rhinitis which we felt was due to their dust sensitivity. We have been treating them with deep X-ray therapy given through the tissues behind the angles of the jaw at biweekly intervals, obtaining a total of six exposures. We have seen no unfavorable effect as far as dryness of the throat is concerned. The lymphoid tissue does not disappear but some atrophy does take place and the inflammatory appearance usually disappears. The acute upper respiratory infections with resulting bronchitis and asthma have been definitely diminished.

We were interested in studying these infective asthmas from the allergic standpoint and especially in their relationship to hyperplastic sinusitis. In the first place, we found that 30 per cent of them showed other allergies, such as hay fever, eczema, migraine, etc.; and secondly, the blood showed an even greater percentage of eosinophilia than did the atopic cases. The blood eosinophilia of atopic asthma has long been recognized but the comparison between the two types had not been made. In 31 infective cases, 19 showed a blood eosinophilia of over 5 per cent, while in 18 cases of atopic type, eight were over 5 per cent. In other words, the percentage of eosinophilia was more marked in the infective cases. Thirty-two cases of the combined type showed over 5 per cent in 24 cases. We also examined the antral discharge in 16 of these infective cases and 13 had an eosinophilia of 3+, while allergic rhinitis cases with infected antra showed a 3+ eosinophilia in six cases. These observations seem important to us in determining the allergic nature of these infective cases.

Our bacteriological findings in these hyperplastic sinusitis cases are most interesting and instructive. We set out to determine if negative sinus cultures meant a negative membrane culture and also if the organism grown from the washings was the same as that in the membrane. We used 34 operative cases for our studies—19 ethmoidal and sphenoidal and 15 antral cases. Some of these cases had been followed for as long as eight years and cultured frequently during the acute and chronic stages. These antral washings were obtained by using a Coakley irrigator. In some cases direct swabs were taken from the antrum at the time of radical operation. All were cultured on blood agar and beef infusion broth. The membranes removed at operation were taken to the laboratory immediately, the surface sterilized, the tissue ground up and then cultured. We were greatly surprised at our results. Fifty-three per cent of the cases showed one or more organisms different from those ever obtained from the washings; 35.3 per cent were the same organism and 26.4 per cent were negative. These percentages are not based on 100 per cent, as some cases showed the same and different organisms. Eight of the nine negative cases were from ethmoidal polyps, which coincides with the general consensus of opinion that these isolated polyps are usually sterile. The importance of recovering a different type of organisms from the membrane is realized when we state that they were usually a hemolytic staphylococcus aureus or the hemolytic streptococcus—alpha or beta types. We also had these membrane specimens examined pathologically and 62.5 per cent of them showed definitely demonstrable organisms in the tissue, mainly cocci. Kistner, at the University of Oregon Medical School, has done some work of a similar nature. We feel, as does he, that the hyperplastic sinusitis is of bacterial allergic origin and are making some progress through the use of autogenous vaccines from these membranes in demonstraitng this nature.

DR. PEER M. LUND: I should like to show some slides demonstrating sinus conditions, which we in the past have been accustomed to diagnose and deal with as purely infectious changes, but which we now have learned to recognize as *allergic reactions*. They are probably *manifestations of general hypersensitiveness* in the *individual*.

Before the generalized use of lipiodol and brominol injections into the sinuses, the X-ray diagnoses were at best rather vague but now we are really to a certain degree able to make quite definite statements as to the causes and intrinsic nature of "cloudy antra and ethmoids."

In the sinus conditions appearing on an *allergic* basis, we have to do with an edematous membrane of greater or less thickness and smoothness; the more acute the condition the smoother we find the contour of the opaque shadow, while in cases of longer standing where inflammatory changes have set in, we find the opaque shadows smaller and irregular in contour; also in cases where the edema of the mucous membrane involves the ostium we find that no fluid enters the sinus.

AMERICAN OTOLOGICAL SOCIETY.

The Sixty-fifth Annual Meeting of the American Otological Society, Inc., convened at Hotel Chelsea, Atlantic City, N. J., May 17-18, 1932, at 9 a. m., Dr. L. W. Dean, of St. Louis, presiding.

Presidential Address. Cytological and Bacteriological Studies of Otitic Disease. Dr. L. W. Dean.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Report of the Committee on the Study of Progressive Deafness.

DR. ARTHUR B. DUEL reported that the economic depression had influenced the income of the research fund. He mentioned the allocation of funds and stated that Dr. Wilson would present details regarding the committee's report.

DR. JOHN GORDON WILSON: The report of your Committee on Progressive Deafness, which I now present, this year is chiefly confined to the work of Dr. Best, of Dr. Crockett and of Dr. Davenport. It is to be noted that satisfactory investigations are under way at McGill University with Dr. Tait; at Illinois University with Dr. Culler, and at Northwestern University with Dr. Anson. Next year we expect to give a full account of this work.

DR. BAST'S report on the development of the petrous temporal bone:

1. To date we have serial sections of a representative chronological series of 50 human fetal ears.

2. We have collected seven ears from young children, some of which are ready for sectioning.

3. We have constructed 14 different models of human ears showing growth of the capsules and blood supply.

4. The following papers have been published on the ear: a. 1928—The Utriculo-Endolymphatic Valve. b. 1928—Ossification of the Labyrinthine Capsule. c. 1929—Osteogenesis of the Human Periotic Capsule. d. 1930—A Comparative Study of the Utriculo-Endolymphatic Valve in Some Common Animals. e. 1930—Ossification of the Otic Capsule in Human Fetuses. f. 1931—Blood Supply in the Otic Capsule in a 150 m.m. (C.R.) Human Fetus.

A seventh paper, on the "Resorption of the Cartilage in the Canal Portion of the Otic Capsule in Human Fetuses and Its Relation to the Growth of the Semicircular Canals" (nine plates and 34 figures), has been accepted for publication in the *Archives of Otolaryngology*.

It is now possible to come to some tentative conclusions regarding the question whether the otic capsule is predisposed to subsequent change. In the metamorphosis of cartilage to bone the normal tendency in most bones is completely to replace the cartilage by a primary endochondrial (geflechtartig) bone and this into a secondary or lamellar bone, sometimes directly from cartilage into a secondary bone. Primary endochondrial (geflechtartig) bone is always present in bone and represents a stage in bone formation. It is normally removed by vascular buds and replaced by lamellar bone around the vessels, except small remnants which remain between the lamellar bone, the so-called interstitial bone. In the otic capsule, however, the bone formation is an incomplete one. In the first place, in small isolated areas which present individual variation in number and distribution, the cartilage matrix is not completely removed; only the lacuma of such cartilage is changed into primary bone while the cartilage matrix persists in calcified cartilage. Such areas are known as interglobular spaces, cartilage islands, intrachondrial bone. Between these areas an endochondrial (cartilage) bone is formed, in character ranging from a primary (geflechtartig) bone to a secondary lamellar bone.

DR. CROCKETT'S report on the influence of body calcium on otosclerosis: The influence of increased body calcium in the etiology and on the progressive nature of otosclerosis has been much in dispute. To study this subject a grant was given in 1927 to Dr. Crockett and Dr. Aub, of Boston. It was arranged that the early and more intensive part of this investigation should be carried

on in the metabolism wards of the Massachusetts General Hospital and that later, when the patients returned to their homes, they should still remain under observation and guidance. This plan has been successfully carried out. The investigation, therefore, has been in progress for nearly four years. In all, 23 cases have been studied. In 1927, for one month they were strictly confined to the special metabolism ward under Dr. Aub's supervision on a carefully prepared low calcium diet, with a special dietician. The food intake was carefully weighed and was adequate in everything except calcium. The output of calcium in the body secretions was carefully measured.

Conclusions: 1. No marked improvement in hearing can be expected in any case where the diseased process has existed much over a year. On the other hand, where the otosclerosis has not existed over a year and where the amount of bone change as judged by the hearing test is not great, an improvement may be effected. In three cases, none over 12 months' duration, the improvement in hearing was marked; the hearing had become normal (audiometer reading), though the tinnitus had not quite disappeared. This improvement has been held to date, now two and a half years.

2. A slowing of the progress, it may be a possible cessation of the disease, may be looked for in older patients where the process is of longer duration, by the use of the diminished calcium intake diet of the type previously mentioned. Such a diet is easy for any patient to carry out.

3. While the use of parathormone to pull calcium from the body greatly assists in calcium output, its use has been found unnecessary. It is very expensive and to be administered necessitates the patient to be hospitalized. The same result can be obtained in a simpler, slower way by the administration by mouth of 50-60 grains ammonium chlorid once a day, limited to their tolerance.

Dr. Davenport's report on the genetical study of otosclerosis: The study of the influence of heredity on otosclerosis was undertaken by Dr. Davenport, of the Carnegie Institute of Genetics, Cold Spring Harbor, Long Island, N. Y. Dr. Davenport directed his investigations to an analysis of the factors responsible for the development of this defect, particularly the genetical factors, especially using the method of Mendelian analysis.

Conclusions: The hypothesis of two dominant genes is not negatived in any of the groups of matings. The hypothesis that otosclerosis depends on a single dominant factor, a single recessive factor, a sex linked recessive and dominant autosomal gene, two recessive genes (one sex linked), also on a dominant recessive and a recessive autosomal gene, all meet with difficulties in application. The conclusion is reached that the simplest formula that approaches agreement with the findings is that of two dominant genes, of which one is sex linked.

No geneticist believes that the genes which shape the construction and constitution of our body constitutes the whole story of the transmission of a morbid condition or a malformation. Characteristics are not transmitted as such from parents to offspring. What is inherited are hereditary units which interact and give the plant or animal the power to respond in a certain way to a given kind of environment. The genes determine the possibilities, the actual realization of any possibility is dependent also upon environment on hereditary factors. Environment is more potent on the results when acting on a suitable individual. The fact that a smaller percentage of otosclerosis occurs in some families than in others appears to show that influences other than the gene are at work.

Is All Hearing Cochlear? Dr. John Tait.

Our VIIIth cranial nerve subserves both an exteroceptive (auditory) and a proprioceptive (equilibrium) function. Its equilibrial receptors, anteriorly placed, are the semicircular canals and the utricular macula. The cochlea, posteriorly situated, is auditory. Meantime the function of the saccular macula which lies between the proprioceptive and the exteroceptive sense organs is unknown. The author marshals evidence in support of the view that it is auditory.

While the cochlea and its accessory apparatus, all of light construction, vibrate in an otherwise immobile head, the saccular macula presumably responds to actual vibration of the head. Some of the otolithic receptors of aquatic

invertebrates respond to water-conveyed tremor. In lower vertebrates there are at least two otolithic receptors in the posterior part of the labyrinth, namely, the lagena and the saccular macula. G. H. Parker has obtained evidence that the saccular macula of a fish is auditory. Mr. D. A. Ross, a worker in the author's laboratory, has shown that both the lagena and the saccular macula of certain fishes are auditory.

Strangely enough, the croaking mechanism of frogs is designed not primarily to transmit air-borne sound, but for the purpose of causing water tremor, guided by which the sexes are brought together. Some fishes similarly use their air bladder for sexual signalling under water. The voice of vertebrates took origin at an early stage, and then for sexual purposes it began in relation to the air bladder of fishes. This air bladder became transformed into the lungs of terrestrial descendants. By the transformation the tremor remitting characteristics of the original air bladder were not sacrificed but, being effective also for production of air-borne sound, were retained as the basis of voice in all the higher vertebrates.

The author holds that it is especially during voice production that the saccular macula is stimulated. It is thus a proprioceptive and not necessarily an exteroceptive auditory organ. It responds to the head tremor that inevitably accompanies spoken voice. Only when we whisper can we hear our own voice by cochlea alone. An "R" sound cannot be whispered, because it involves a vibratory trill. It is because of the absence of saccular stimulation that one's own voice as reproduced by phonograph fails to be recognized.

DISCUSSION.

DR. J. GORDON WILSON: The saccula has gone through many vicissitudes; one thing is clear, that the role it has been supposed to play in equilibrium will either have to be abandoned or greatly modified. All evidence at present points to its importance as a receptive mechanism for what may be called gross vibrations—an interpretation which Dr. Parker some years ago suggested as a result of his experiments on fish. Dr. Parker's results Dr. Tait has confirmed in the frog and amplified in great detail. The clinician thinks back on those puzzling cases where with good evidence that the cochlea was destroyed, the patient seemed to pick up vibrations with extraordinary ease.

In his discussion of the relation of the larynx to lung development, Dr. Tait raises an important problem; in how far was the lung developed as an adjunct to the larynx and emotional reactions; in how far was the larynx developed in a position where vibrations could be easily produced? While it appears that the mass of evidence supports the latter hypothesis, it may well be that both have to be taken into account.

MR. R. L. WEGEL: Prof. Tait has reported an interesting demonstration which indicates that in lower animals the sacculus and utriculus are sensitive to vibration and that their excitation constitutes all or part of the "hearing" in such animals. The thought suggests itself that in these animals the threshold referred to as "feeling" in man is lower than that of hearing, if indeed hearing as in higher animals exists at all. There are at least two measurable thresholds in the human ear, auditory and "feeling," and at least six possible thresholds. It is to be taken for granted that any one of the nerve terminations in the labyrinth—three ampullae, sacculus, utriculus and organ of Corti—may be excited by sound vibrations if loud or violent enough. Some observers describe a sensation of dizziness at the "feeling" threshold, suggesting excitation of one or other of the semicircular canals. The sacculus and utriculus appear to be more favorably situated for picking up vibrations, which circumstance gives color to the hypothesis that they are responsible for the hearing threshold at low frequencies. There is against this the fact that the hearing threshold is continuous from the highest down to the lowest perceptible frequency and the high frequencies are without doubt perceived in the cochlea. Prof. Tait's paper is valuable not only in the physiology of lower animals, but also in bringing attention to the likelihood that the sacculus and utriculus, as well as the cochlea, are excited at low frequencies in man.

DR. EUGENE R. LEWIS: As Mr. Wegel was talking I recalled a patient who complained of a peculiar nausea every evening, at the same time, during the summer. This was not associated with vomiting or pallor. At this hour, bats

were flying from the nearby church belfry. The bat's squeak is a supertonic tone, and this may have irritated the chorda tympani, causing not only nausea, but syaluria.

MR. R. L. WEGEL: By increasing sound intensity you can arrive at a point where people complain of feeling dizzy or are nauseated. This suggests that these vibrations affect the semicircular canals. The saccular macula being more favorably situated has received these vibrations, which brings up the interesting point of hearing by other means than the cochlea.

DR. STACY R. GUILD: These views are more or less historical. Corit, Henson and Helmholtz, among others, thought that the cochlea served only for analysis of tones, and that the maculae and cristae served to mediate other types of vibration. Hensen and Lucae, in the first decade of this century, clung to the view that the vestibular end organs also have auditory function. So far laboratory examinations of macula sacculi in cases of total deafness have not given anything of interest, but with Dr. Tait's work in mind I am going to give the problem more attention, especially in cases where discrepancy exists between the amount of the loss of hearing, and the microscopic pathology of the middle and inner ear.

DR. EDMUND P. FOWLER: The old theory was that the cochlea was for the differentiation of sound, and the other structures detected noise; Dr. Tait's work seems to be proving this. Perhaps the irritation or stimulation of the saccule disturbs the cochlea function, just as any noise will mask any other noise, or as tinnitus interferes with hearing. This research suggests that noise tinnitus comes from inflammation of the saccule, and not from the cochlea at all.

DR. LORENTE DE NO: Years ago, I made some experiments on ablation of the saccular macule in the rabbit. I found no spontaneous symptoms. The labyrinth reflexes of the eyes seemed to be quite normal, but when the rabbit was turned on the turning table and the eye reflexes recorded there appeared at the same time as the nystagmus slow changes in tonus of the eye muscles: that, of course, represented slow oscillations of the eyes. I do not regard these experiments as conclusive because in order to destroy the saccular macule it is necessary to open the membranous labyrinth, and the perilymph and endolymph have a quite different pressure after the operation. We may say then that until now there is no physiological evidence of any participation of the saccular macule in the regulation of the equilibrium and allied function. But anatomically, the evidence is strongly in favor of the view that the saccular macule must have a function very similar to that of the macule of the utricle and quite different from that of the cochlea. I have been able to follow in histological preparations stained with the silver methods, the nerves of the saccular maculae, into Scarpa's ganglion and thence into the medulla, where the endings are partly in common with the nerves of the utricular macule. On the other hand, the cochlear nerve has nothing in common with the saccular nerves. We must also think that the saccular macule has a function related to that of the utricle and semicircular canals, and independent of that of the cochlea, but of course nothing can be stated until physiological evidence is available.

DR. JOHN R. PAGE: I reported a case of paracoustic vertigo some years ago in which sound produced a movement of the eyes and a tendency to fall in the plane of the right anterior vertical canal. The hypersensitiveness in this canal became so disturbing that the patient could not speak above a whisper without falling. Finally relief was afforded him by operating on the labyrinth. The vestibular and cochlear regions were opened and the anterior vertical and horizontal semicircular canals were removed.

PROF. TAIT (closing): Dr. J. S. Fraser once told me that by the older physiologists the saccule was associated with the cochlea. In lower vertebrates a number of different receptors occur in this auditory part of the labyrinth. We must keep in mind the distinction between response to noise and reception of tone. Some of the primitive auditory receptors may be for noise, others for tone. In answer to Dr. de No, stimulation even by the utricular otolith is not constant. The muscles even of a delabyrinthized animal can hold posture.

When, by a tilt of the head, the utricular otolith slips, the utricle changes a set posture. Both utricle and saccule may be somewhat analogous in their mode of stimulation. Dr. Wilson has found a valve between the membranous saccule and the membranous utricle in mammals. To discriminate between the different auditory receptors in the frog, we shall have to examine the animal in its natural surroundings in the spring time. We shall then combine laboratory with field experiments.

Physical Measurements and the Physiology of Excitation of the Auditory Nerve. Mr. R. L. Wegel.

Practically all phenomena of hearing studied by so-called physical measurements involve as an essential component certain reactive properties of tissue. In order to discover the details of the physical mechanism from information so obtained, it is therefore necessary to know the properties of the tissue of the nerve tract as a meter of stimulus or in the absence of such knowledge to be content with the best obtainable approximations. This paper deals with the interpretation of certain data on excitation of tissue into a form applicable, as nearly as possible, to the problem of hearing.

The diffusion theory of excitation of Nernst provides a quantitative method of calculation of excitation occurring within a time interval of three chronaxie. For longer times the incidence of changed metabolic rate modifies the laws. Latent additional data of Chauchard indicates an increase in excitability by a factor of ten or more. The indications of discrimination data are that the auditory nervous tissue responds in this supernormal phase near the threshold.

Absolute pressure acuity at low frequencies varies inversely as the cube of the frequency, indicating that the end organ here is responsive at threshold to its mechanical acceleration. Stated in other words, the velocity of motion required by the basilar membrane for threshold excitation is proportional to the time interval between successive impulses in the sound wave when this interval is long, compared with the chronaxia of the nerve.

An important factor in binaural hearing is the cross-connections between the two sides of the auditory nerve tract. From observations on binaural beats by Stewart, it is concluded that the time interval required for this cross-propagation is from .010 to .001 seconds, depending on frequency, in the normal ear. The phenomenon disappears, as frequency is increased, at about 1,000 cycles, presumably because of the statistical nature of nerve impulse generation, resulting in part from the encroachment of stimulus on the refractory phase of the tissue.

DISCUSSION.

PROF. K. S. COLES Mr. Wegel has emphasized the complete chain of mechanism involved in hearing. His data suggests that in the range below 1,000 cycles the mechanical system from the outer ear and the nervous system to the brain are both capable of transmitting subthreshold intensities, but the sensory end organ is the weakest link in the chain. As shown by Adrian, the problem of the sensory end organ is an outstanding physiological one at this time.

If we assume that acceleration of a hair will bring the sensory cell into activity, then at frequencies above 1,000 it may be stimulated without impulses reaching the brain, since another mechanism, the nerve itself, is responsible for the behavior of the entire. The nerve, however, is a problem by itself.

The work of Galvani, in the eighteenth century, was the forerunner of the birth of physical chemistry. Recently Nernst gave to physiology his theory of nerve stimulation. Mr. Wegel's work is both stimulating and instructive. It suggests that the most important problems of hearing are those physiological problems which are being powerfully attacked at the present time—sensory end organs and nerves.

PROF. TAIT: Mr. Wegel has utilized the latest studies in work respecting nerve conduction and the way in which he has done this shows what a good physiologist is hidden in the Bell Telephone Company. He takes the subjective sensory findings as determined in his own laboratory and attempts to explain these in the light of the most recent physiological data. He employs mathematical analysis to explain the happenings within the cochlea. This side of

his paper I am incompetent to discuss, and I think it is a striking thing that papers of this type should be presented here.

DR. ELLISON ROSS: The transmission of the nerve impulse is a common physiological process — we have metabolism oxidation, and reduction, elimination of energy, fatigue and recovery. Another factor, however, that is disturbing, enters into the problem; that is electricity. A nerve trunk is a bundle of fibres more or less electrically insulated from each other. The general plan of the nervous system is that of a common commercial electrical system. The Wever-Bray phenomenon with the auditory nerve argues strongly for the importance of electricity in nerve stimulation. Electricity is the great problem still ahead. Mr. Wegel has employed highly mathematical and physical methods of attack which will constitute a great advance in this field. However, we must not draw too general conclusions. Living tissue-chemistry, temperature, blood supply, toxins and the colloidal state have their influence. Small changes in any of these factors may make very great differences in the composite result in a nerve.

DR. EUGENE R. LEWIS: One factor that seems worthy of consideration is inertia. To *initiate* activity, whether in one cell or a combination of cells, may require more force than is required to *maintain* activity. Ultimately, increasingly intense stimuli are followed by fatigue or exhaustion of the cell. In some types of nerve mechanism there seems to be some self-contained factor which makes for continuation of action until cessation or fatigue occurs. As the store of available energy for response approaches the exhaustion point, the cell shows physical changes in form and size. A final stage, cellular dissolution, is reached when opportunity for restoration is denied to the cell. One sees evidence of maximal, minimal and pathological degrees of response.

PROF. DAVIS: The slow-acting tissues, such as the smooth muscle tissue of the alimentary canal and of blood vessels, we can think of as the opposite extreme from the ear. I conceive of the ear as having extremely rapid action. The impulses that follow the auditory pathways have frequencies up to 900 vibrations per second. It is in this region that a discontinuity occurs and I suspect that there is significance in that coincidence.

The activity of the sense organ is divided into the fast and slow types. The slow organs are stimulated by continuous excitation, such as tension or temperature. In the tactile cells, where the discharge is confined to the moment of contact, there is rapid adaptation of the sense organ to the stimulus. The cochlea, as Dr. Tait suggested, is a modified tactile organ. Where the conditions are favorable, for the transmission of sound waves to the auditory nerve, it is possible for the impulse to follow at fairly high frequencies, the limit being imposed by the recovery of the nerve itself.

MR. WEGEL (closing): In regard to the discussion by Profs. Cole, Tait and Ross, I wish to express my gratitude to them for having passed over many of the obvious faults of the preliminary manuscript which they had for reading and for having constructively discussed those points which were intended as contributions. I feel very strongly, as does Prof. Cole, that much that is without doubt essential can be learned about hearing by a further and more discriminating study of the more easily handled tissues of lower animals. Prof. Davis points out the uncertainty of quantitative results obtainable from Chauchard's data. This question is also raised from still another angle in the manuscript, and I should like to point out that the object has been to introduce to ear specialists the possibility of dividing the time of reaction of a tissue into a physical and a metabolic phase, and to describe an approximate method of calculating the first. This concept may be applied to any measurement. Dr. Lewis' question concerning mechanics of hearing leads directly into a rather copious philosophy which cannot be adequately treated in a short discussion. This question has been considered in a number of past meetings and printed in the Transactions.

The Relationship of Upper Respiratory and Alimentary Tract Flora to Mastoid Infections, With Particular Reference to the Epidemiology of Mastoiditis. Dr. Samuel J. Kopetzky.

Printed in full in the September issue of THE LARYNGOSCOPE.

DISCUSSION.

DR. J. G. DWYER: In regard to Dr. Kopetzky's work, our results in the main tally with his, although we have not done such careful subculturing. Of course we all recognize that the hemolytic organisms play the major role in these middle ear and mastoid infections and in their ensuing complications, and also that there are certain years and certain months in the year when these infections seem to be greater in number and in severity. We have been accustomed to attribute this to increased virulence of the organisms in different epidemics, which in the main seems to be true, although Dr. Kopetzky's classification tells us that it is due to the different types of streptococcus. I can see that this subculturing will give us definite information for indications for operation, as well as for prognosis.

DR. EDMUND P. FOWLER: Not being a bacteriologist, I should like to inquire how it is possible to obtain cultures from the chronic discharges that do not show many forms of organisms. From the mass of material, how do you pick out a certain type and say that this organism is accountable for the chronic discharge?

DR. KOPETZKY (closing): I will answer Dr. Fowler first. The bacteria were taken from the middle ear at operation. When this was opened, we took a swab from a sterilized tube and wiped off the secretions. In the ensuing growth, saprophytes and contaminations were cultured, and only streptococci transplanted for culture.

In regard to the classification, it would be a very desirable thing if studies similar to ours were instituted in different sections of the country. We should then have a more complete survey of the incidence and mortality rates by competent observers all over the country.

Circumscribed Arachnoid Cyst Giving Symptoms of an Acoustic Neurome.

Dr. William V. Mullin.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Two cases, which at operation were proven to have cystic tumors of the arachnoid, were studied at the Cleveland Clinic. A review of these two cases and of others in the literature has raised the questions in the author's mind as to whether it might be possible to make, before operation, a diagnosis of cyst of the arachnoid from the history and a complete and thorough examination of the ears. There is not much correlated data in the literature.

A very complete history, making a point of ascertaining the sequence of symptoms, together with a complete vestibular and cochlear examination, might do much to make possible a better diagnosis. There may be a variance in the age groups of patients with acoustic tumor, and those with arachnoid cysts. Infection and injury may both be etiological factors. Duration of symptoms may also be important.

In the first case studied there was some hearing in the left ear (the diseased side), with prolonged bone conduction on that side, with no responses from both horizontal canals and with no eye changes. If I were to encounter again a patient with such bizarre symptoms I would be suspicious of disease of the arachnoid.

In the second case, there had been sudden onset of symptoms, namely, vertigo, vomiting and tinnitus with complete remissions; spinal puncture completely relieved all the symptoms once; eye grounds and visual fields were normal; there was no spontaneous nystagmus; some responses remained to caloric stimulation on the right side with normal responses on the left. This ruled out acoustic neuroma and while it seemed at the time that degeneration of the right VIIIth nerve might account for the symptoms, yet the history of the onset of symptoms while stooping, the severe vertigo followed later by tinnitus, and the fact that two years after the onset of symptoms a spinal puncture gave entire relief from all symptoms for five months might have rather definitely ruled out such a diagnosis, and might have aroused suspicion of the presence of an arachnoid cyst.

From the present meager data, I believe that it is impossible to form any conclusions, but it might be that further study would elicit information which might make possible a differential diagnosis in these cases. The subject has

been given practically no consideration by otologists, and it would seem that there has been a decided neglect on the part of neurologists and neurologic surgeons to have complete functional ear examinations made before operation.

DISCUSSION.

DR. LEWIS FISHER: An experience with a large number of cases with brain lesions demonstrates more than ever the difficulties of intracranial diagnosis. Among the many pitfalls encountered, that of arachnoid cyst is by no means the least important, and Dr. Mullin in calling attention to this lesion is offering a timely contribution. While arachnoid cysts are apt to lead the examiner astray in many and various types of intracranial lesions, the author's choice of cases with the lesions really otologic in nature adds greatly to the value of his discussions. One cannot emphasize too frequently that intracranial lesions involving the cerebellopontile angles belong particularly to the field of otology, and that the responsibility of a correct diagnosis rests directly upon our shoulders, and not upon those of the neurologist or the neurosurgeon. You will recall that in one of the cases presented by Dr. Mullin (not his own case), a submucous resection had been performed in the attempt to relieve the patient of his deafness and tinnitus, before the true nature of the lesion was discovered. With the essayist, I feel that it is unfortunate that a complete examination of the function of the labyrinth, both cochlear and vestibular, is not performed as frequently as it should be.

DR. MARVIN JONES: A caution should be uttered against advising lumbar punctures in lesions of the posterior fossa because this would mean exerting force on the medulla and might cause respiratory paralysis and death. A preferable procedure would be to trephine the skull and aspirate the lateral ventricle.

DR. STACY R. GUILD: Cystic formations are frequently found in association with acoustic tumors. In two cases in my practice the tumors were so located in the internal meatus that they could not possibly have been seen during operation. In view of these facts, I would like to ask Dr. Mullin whether in the cases which he has called arachnoidal cysts simulating cerebellopontile angle tumors, he can be certain that there was not actually a small tumor in the internal meatus with secondary cystic formation.

DR. J. GORDON WILSON: The differential diagnosis between a neoplasm at the cerebellopontile angle and a cystic formation in that area, in my experience is extremely difficult. The symptoms presented are more or less common to both. This is not to be wondered at since they are both largely traceable to pressure.

The symptoms of a cystic development are best understood from a study of the anatomical relation of Luschka's foramen and the prolongation through it of the choroid plexus from the fourth ventricle, to the surface of the cerebellum and the nerves emerging from the pons and medulla (slides shown illustrating this relationship from Traite d'anatomia humaine publie par Poirier et Charpy, Paris, 1901, systeme nerveux III—1, pp. 133, 152). With these anatomical relations it is easily understood how a localized subarachnoid cyst may be formed and how it can involve not only the VIIth and VIIIth nerves but the VIth, IXth and XIth.

DR. W. V. MULLIN (closing): Dr. Fisher has given us in the past something so very definite on which to base a diagnosis of tumor of the cerebellopontile angle that when in any case the findings do not correspond to the symptomatology that he has given us, it is sometimes hard to convince the neurological surgeon that there is disease in this region other than an acoustic tumor, and that the patient should be explored. I am observing a case now, but several surgeons hesitate to operate because the typical angle syndrome is not present, and because the deafness is of the conduction rather than of the perception type. This particular phase of it is merely a coincidence. If they do not operate soon it will be too late, for the man will be killed driving his automobile, which he persists in doing despite severe attacks of vertigo.

In conclusion, I believe patients with brain cysts apparently have a protein symptomatology, with considerable variation in hearing and labyrinthine response.

Vidian Neuralgia, With Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Apex. Dr. Harris H. Vail.

In this paper is raised the question whether the ocular and periorbital pain so typical in cases of suppuration of the petrous apex is due to an irritation of the ophthalmic division of the Vth nerve. The recent literature is reviewed showing that the opinion is held by most authors that his typical ocular pain is due to an irritation of the first division of the Vth nerve. Search of the literature also fails to reveal any particular significance being given to the great superficial petrosal nerve. Anatomical studies show very close relation between the great superficial petrosal nerve and any pneumatic cells in the anterior portion of the petrous bone.

Three typical cases of vidian neuralgia due to sphenoid disease are reported to show the great similarity between the eye pain in this condition and the eye pains in suppuration of the petrous apex. The author concludes that:

1. Anatomical studies show the very close relation between the great superficial petrosal nerve and any pneumatic cells which might be present in the anterior portion of the petrous bone.

2. It has also been shown that impulses coming over the great superficial petrosal nerve can reach the orbit and from there pass over the terminal branches of the ophthalmic division of the trigeminus by means of the anastomosis between these nerves and the orbital branches of the sphenopalatine ganglion.

3. Three cases of vidian neuralgia are reported to show that irritation of the vidian nerve in the floor of the sphenoid sinus causes eye and orbital pain.

4. The eye and orbital pains described by patients with suppuration of the petrous bone show a great similarity to those found in cases of vidian neuralgia.

5. The great superficial petrosal nerve is extradural throughout its course. The Gasserian ganglion and its divisions are above the dura. Hence, there must be a dural involvement before the Gasserian ganglion and its branches are affected.

The question raised at the beginning of the article is answered as follows. It would seem that the eye and orbital pains so typical of petrous suppuration in the early stages are not due to an irritation of the ophthalmic division of the Vth nerve, but are produced by the irritation of the great superficial petrosal nerve caused by the suppuration in the petrous bone.

DR. D. E. WISHART: I am interested in disease of the petrous apex. I have two patients suffering from multiple osteomyelitis. In one patient, hearing is lost in both ears and the vestibular responses have disappeared. The second patient has lost entire function of the VIIIth nerve on one side and on the other side he has partial loss of hearing with partial loss of vestibular response. The suggestion is that the focus of infection lies in the petrous apex. In the absence of middle ear signs, how can this be diagnosed? Has Dr. Vail in his study of disease of the petrous apex come across any cases similar to mine?

DR. VAIL (closing): When Dr. Eagleton said he would discuss my paper, I knew he would bring out very important facts. He has gone to a lot of trouble to look up these cases and I am very grateful to him. It is obvious that we must diagnose suppuration of the petrous apex early, and the significant pain back of the eye in these cases is a symptom which appears early. After all, it does not matter a great deal just how the pain is produced, so perhaps my paper may be splitting hairs. The important thing is what Dr. Eagleton has mentioned, namely, the significance of the pain back of and around the eye on the same side as the affected ear.

I am unable to answer Dr. Wishart's question. The labyrinthine disturbance may have been due to either a metastasis from the infection, or a toxemia.

Treatment of Otitic Leptomeningitis. A Plea for Investigation in Two Directions: 1. Kubie's Theory of Forced Drainage; 2. Direct Surgical Drainage. Dr. Philip D. Kerrison.

This outline of new methods on the treatment of otitic meningitis has been presented because the surgical treatment of otitic leptomeningitis has produced only meager results. We still lack a rational, clearly defined and generally

accepted plan of surgical treatment which, in any large series of cases, can be depended upon to save even a small percentage of lives. E. W. Day, in 1913, after an exhaustive study of this disease concluded that, "theoretically, surgery should give relief; practically, the mortality has not been changed by it."

In passing on to the most recent experimental work by Weed and Kubie, the author pauses to criticize the work of Eagleton on subarachnoid irrigation; on withdrawal of spinal or cerebrospinal fluid, with replacement with Ringer's solution; and also on ligation of the common carotid artery, as procedures lacking a physiological basis. He prefers to advocate therapy in the direction of forced drainage, or of direct surgical drainage, according to the studies of Kubie and Weed.

A brief description of these methods is given: Kubie's theory of forced drainage: The cerebrospinal fluid is described as a dialysate, or filtrate, from the capillaries of the choroid plexus, and in small amounts from the perivascular and perineural spaces of the central nervous system. Due importance is given to this latter source of the filtrate.

Further, the cerebrospinal fluid is stated to be in osmotic equilibrium with the blood. Injection of distilled water, or hypotonic solution or drinking large amounts of water causes increased intracranial pressure, and increase of spinal fluid production. If, however, hypotonic solution injection permits loading of the blood with salts, osmosis works the other way, and results in diminished brain bulk, and diminished cerebrospinal fluid production. Thus intracranial pressure and cerebrospinal fluid production may be changed at will in either direction by dilution or concentration of the blood salts.

Weed, in 1923, experimented on dogs, in order to determine the source of the cerebrospinal fluid. He found it to be in the choroid plexuses and perineural and perivascular spaces of the brain.

Kubie's work was based upon the therapeutic possibilities of Weed's experiments. He worked first on dogs, and instituted continuous drainage from the cisterna magna, making pressure observations through a trephine hole in the parietal bone. The subarachnoid space was not entered. The cisterna magna was connected with a second manometer, and by this means pressure could be recorded either when there was a flow from the cisterna magna, or when it was shut off. Pressure changes varying from 2 to 5 m.m. were observed with each heart beat; greater changes caused by inspiration and expiration; pressure reduction during outflow from the cisterna; and pressure changes resulting from injections of hypotonic solutions.

Kubie states that in inflammatory conditions of the central nervous system and of the meninges, the perineural and perivascular spaces are lined with newly formed cells, many of which are lymphocytes. Following intravenous injections of hypotonic solutions accompanied by continuous drainage of the cerebrospinal fluid, masses of these cells may be seen at postmortem to have been extruded into the subarachnoid space.

Further, dogs upon which these experiments have been repeatedly made and at intervals of only a few weeks have made apparently permanent and perfect recovery.

The basis of the theory then is that the perineural and perivascular spaces, which, during meningeal inflammation are the supply posts for the enormous increase in the number of lymphocytes, are also the storage points of pathogenic micro-organisms, and that since these cell-filled and infected crevices are in part the normal starting points of fluid production or dialysis, a stimulated increase in fluid production may rid the central nervous system of toxic matter through an autogenous cleansing of its perineural storage points. It is urged that a group of men of scientific attainments and technical skill, working in a well equipped laboratory, should further elaborate this work in its therapeutic aspects.

(To be continued.)

DR. SIDNEY YANKAUER.

IN MEMORIAM

DR. SIDNEY YANKAUER.

The ranks of the Old Guard are thinning and the most recent to be called is Dr. Sidney Yankauer, of New York. He died of heart disease, Aug. 26, 1932, at Mount Sinai Hospital, New York, the institution in which he had served as laryngologist since his graduation from the College of Physicians and Surgeons of Columbia University, New York, in 1893. He was also consultant laryngologist at the Beth Moses Hospital, Brooklyn; the Montefiore, Broad Street, Beth David and Beth Israel Hospitals, and at St. Joseph's Hospital at Far Rockaway, L. I.

He was a pioneer in the field of bronchoscopy and contributed much toward its development as an organized specialty in this country.

During the World War he served in the A.E.F., as a Major in the Medical Corps.

His contributions to the treatment of lung abscess by bronchoscopy, inspection of the nasopharynx by a specially devised nasopharyngoscope, a modified operation for resection of the turbinate, the adaptation of a new type of ether mask and the method of administering anesthesia by regulated compressed air, the use of suction during operations in laryngology, were individual and outstanding.

From 1905 to the time of his death Dr. Yankauer had been a member of the collaboratorial staff of THE LARYNGOSCOPE, and many of his valued contributions to laryngological literature have been published in this journal.

He was president of the American Bronchoscopic Society in 1928; a member of the American Laryngological, Rhinological and Otological Society, the American Society for Thoracic Surgery, the American Academy of Ophthalmology and Otolaryngology, the New York Academy of Medicine, and a Fellow of the American College of Surgeons and the American Medical Association.

To his widow, Mrs. Margaret Kearns Yankauer, and the bereaved family we offer our sympathy.

Personally, we recall and respect him for his geniality, democracy, straightforwardness and scientific attainments. M. A. G.

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